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## SYMPOSIUM ON MENINGITIS.

### MENINGITIS OF OTITIC ORIGIN.*

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*Introduction:* The subject-matter of this symposium deals with one of the most important complications with which we are constantly confronted in our work. It stands out prominently in two respects. In the first place, it is probably the complication in the treatment of which we have made the least progress, and in the second place, it is the one that causes us so much anxiety and care, and gets us as otologists into all kinds of trouble on account of its unexpected development. There is probably no other disease that has written such a tragic history in our annals.

It is interesting to note that meningitis was spoken about in very ancient times, and last year while in London, the writer picked up a treatise on the subject, written in Latin, and published before the discovery of America. The treatise to this day is a masterpiece in the description of the pathology as then known. Again, North, in 1811, wrote an excellent article on "spotted fever", the epidemic cerebrospinal meningitis of today.

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In going over the literature of recent years, and in analyzing the reports of cases, especially the cured cases, one is struck by the fact, that what one observer would consider general purulent meningitis, another would throw out an account of some vital flaw in the exact determination of what constitutes diffuse purulent meningitis. Again another source of obscuration is the inaccuracy in the bacteriological identification of the organisms found. These two basic factors are easily accounted for by the fact, that on the one hand, we do not seem to be agreed as to the essentials of what constitutes diffuse purulent meningitis, and on the other hand, by the fact that bacteriology is practically the "baby" of the medical sciences, and hence the terminology is subject to revision. The only reason for mentioning these points, is to emphasize the necessity for clarification in our diagnosis, so that our records will be worth while. This point will be emphasized later. It would be well to mention at this point, that the observations made here are based on a series of 482 cases seen in the last twenty years and with the recovery of only eight cases.

*Classification:* Otitic meningitis can be classified in various ways, depending upon the basis taken for each classification: some bases are quite satisfactory; others not only unsatisfactory, but quite misleading. Some of the classifications are as follows: That based upon the clinical history, is localized and diffuse. Another on the clinical history, is acute or chronic. Based upon etiology, we have idiopathic, traumatic, postoperative, etc. Another classification is primary and secondary; another serous and purulent. Another, and very satisfactory one, is that based on the bacteriological cause, such as tuberculous, syphilitic, streptococcus, etc. Another is that based upon the pathway of entrance of the organism to the meninges, such as tympanic, labyrinthine, etc. Incidentally, we may here say that there are at least fourteen pathways of entrance, anatomically from the otological standpoint alone. If we could get more information in the individual case, as to the pathway of infection, we might be able to handle our cases before a localized meningitis became a generalized one. Another splendid classification is that from the pathological standpoint, including in this our bacteriological and clinical pathological data. For instance, the course of a meningitis due to the tubercle bacillus, or the treponema of syphilis is very different to that to which we are devoting our attention in this symposium.

*Pathology:* From the speaker's standpoint, it is very important that we should have a thorough understanding as to the underlying pathological changes in meningitis. This, of course, necessitates that we know the anatomy and physiology of the meninges. Essentially

from the pathological standpoint both meningitis and brain abscess start out as the same process. It is a thrombophlebitis of the vessels that is the basic factor, and whether this ends up as a localized meningitis, a cerebritis or encephalitis, or as a diffuse meningitis or localized brain abscess, depends upon two widely separated sets of factors. In very rapidly fulminating cases, there may be a direct invasion, without the thrombophlebitis, such as in secondary invasion from the blood stream, or a rapid throwing in of an infection from a local site. Essentially the reaction set up by the meninges, whether it be in the outer or inner layers of the dura, in the arachmoid, or the pia-mater, or brain itself, is a defensive reaction with the purpose of limiting the process, and effecting a cure, by walling off the infection by exudation of lymph, etc., and a pouring out of cellular elements. It is a defensive reaction on the part of the tissue attacked, tending to localization. Hence we have to view the problem from two angles. On the one hand, we have the type of organism, the virulence of the individual organism and the pathway of the infection. On the other hand, we have the defensive factors of the infected subject. What happens in an individual case, will depend upon a balancing up of these factors. For example, contrast a very virulent organism thrown into a virgin field, in which a wall of localized resistance has had no time to form, with that in which the organism is of low virulence or attenuation, and a good wall or resistance has been set up. In the former case, we will get a rapidly fulminating form of acute diffuse purulent meningitis, running a very short fatal course, such as occurs in direct infection of the meninges, or that in which the wall of resistance is broken down, such as when a polyp is snared through the canal and a rapidly fatal meningitis supervenes. From this series in which many cases of the types of the above kinds occurred, let us quote two: A boy, seventeen years old, was in swimming at Coney Island on a Saturday; developed pain the same night; spontaneous rupture of drum. Within forty-eight hours was wildly delirious with all the classical symptoms of acute diffuse purulent meningitis. Operation on the mastoid showed a hiatus in the attice roof, dura markedly inflamed, etc., and death followed quickly. Here is an example of the direct invasion of the meninges, with an overwhelming infection. An illustration of the breaking down of the wall of resistance set up for years is the following: A man 48 years old, had a polyp snared in his physician's office in the morning on the way down to business, and that evening developed all the symptoms of an acute diffuse meningitis, with a fatal issue in forty-eight hours. In other cases, in contrast with the above, the process is localized and the patient recovers. An example of the slowly developing form of

meningitis, is that coming on in two, three, or sometimes six weeks, after an infection. This is the type usually seen with the capsuled organisms, especially No. 3 pneumococcus. These organisms seem to work in a different way from the streptococci, and will be dealt with later.

Hence, we can see that what happens will depend on the one hand on the organism, its type, its virulence and the pathway of infection; and on the other hand, the setting up of a wall of resistance on the infected subject's part. What eventually happens will depend upon a combination of all these factors. It should be clearly kept in mind that nature's protest against the invasion of the meninges takes the form of a protecting plastic exudate, that is essentially localizing in character and protective in its nature, and that if such is occurring and is recognized it is bad surgery to interfere with this localizing process. While dealing with the pathology, it is interesting, from the postmortem standpoint, to study the cellular elements that are so prominent around the blood vessels, especially the zona vasalis of the pia mater. Contrast the plasma cells, the microphages and gitter cells in purulent meningitis with those found, for instance, in tuberculous or syphilitic meningitis. Again, in our clinical interpretation of the pathology of this disease, we must keep in mind that localized collections of pus at the base of the brain or over the vertex, collections walled off by the adhesive process, may give us symptoms of typical brain abscess with various paralyses and ocular symptoms. These localized collections are important from two standpoints: A diagnosis of brain abscess may lead to brain exploration and any surgical measures directed to the relief of the meningitis would likely be inadequate.

*Clinical Course:* Bearing in mind the above short resume of the pathology with the variations shown by the different organisms, with the variations in virulence, with the different pathways of infections, with the resistance of the attacked subject, we can easily understand that the clinical course will depend upon these and other factors. Hence, it would serve no good purpose to go into case after case, but simply to outline the two extreme types and then place all the others somewhere in between these extremes. On the one side, we have the type following a myringotomy or that going through a fissure, or hiatus in bone, or the dislocation of the stapes, or immediately following the removal of an aural polyp, or injury to the dura, etc. Here we have the overwhelming invasion of the meninges, either through the lymphatics, or blood vessels direct with a rapidly fulminating type of infection and death in a few days, hopeless from the

start. The other extreme type, is the so-called ambulatory form of meningitis, due usually to a capsuled organism especially the pneumococci, in which the condition lasts for weeks, and the only symptoms are slight temperature and headaches, and the diagnosis is based upon a very purulent spinal fluid. Between these two extremes lie most of the cases we meet with in our practice. At this point, it is very important to emphasize the fact, that in drawing conclusions as to the efficiency of any form of treatment in prolonging life, we must keep strictly in mind, the fact, that clinically there are wide variations in the length of time the disease runs its course. The last case in our clinic lived thirty-one days.

*Clinical Pathology:* Here the writer makes a plea that as clinical otologists, we ought to have a thorough knowledge as to the variations in our clinical pathological findings and their significances. We ought to be just as conversant with these findings, as we are with the significance of the albumen, sugar, casts, etc., in the urine. The reason for emphasizing these points, is that in this series, we have found several errors in diagnosis and treatment due to lack of appreciation of these points. This applies especially to the blood counts and the examination of the spinal fluid. Regarding the former, the blood counts, it is our experience that in the ordinary purulent meningitis in adults, the blood count usually runs over 30,000 with a very high polynuclear count. Thus in one case there were 55,000 leucocytes with ninety-eight per cent polynuclears. This is not of course always true, but it is so in the vast majority of cases. The only other clinical condition giving this type of blood count, is acute pneumonia. Hence, a high count would rule out erysipelas, sinusthrombosis, typhoid, etc. This observation does not apply so often to children and so characteristically to the capsuled organisms, but it is a good practical point to keep in mind.

The second point to be emphasized under this heading, is the examination of the spinal fluid. This examination should embrace the following: The contrast between the normal and abnormal fluid under five headings: *1*. Physical; *2*. Chemical; *3*. Cytological; *4*. Serological; *5*. Bacteriological.

This is dealt with in any text book, but as regards the last heading, "Bacteriological," the writer would point out several sources of error under this heading. Unless we can appreciate the changes shown by organisms under certain conditions, our bacteriological diagnosis will be at fault, and it is this point, that is to be emphasized. It is due to mistakes in this field that our records of cases are so much at fault. Thus one author will report a case in which the organisms could be

demonstrated in smear, but could not be cultured. Again, two or three organisms one gram positive, and another gram negative, have been reported in the same cases. Again cured cases with cloudy fluids, but no organisms demonstrated have been reported as acute purulent meningitis. Some of these errors are easily understandable from the bacteriological standpoint. A good healthy organism standing for days in contact with antisera and retained bodies in the spinal fluid changes in two ways: It will lose its power of growth and its staining properties. Hence, an organism may show in smear, but not grow unless a large quantity is planted. Again a gram positive organism may appear as gram positive or gram negative, or both, and lead to the appearance of two organisms being present. Time and again the epidemic form has been diagnosed in these cases when it is only degenerated forms of streptococci and pneumococci, etc. Thus we can see how important it is that we should know this branch of the work.

*Query:* What is the definition of acute diffuse purulent meningitis? Does a spinal fluid with bacteria, etc., as outlined under chemical pathology constitute meningitis? Does a cloudy fluid alone without bacteria justify us in a diagnosis of diffuse meningitis? Does a negative fluid exclude meningitis? What is serous meningitis? Meningismus? The literature is full of case reports, with cures that unfortunately have some flaw in laboratory findings that vitiate the results.

The writer confines the term acute diffuse purulent meningitis to those cases that run the clinical course of meningitis, and in which the organisms can be demonstrated and grown from the spinal fluid. A cloudy fluid alone does not justify us in the diagnosis as to the general form. It is our belief that cloudy fluids are due in these cases to a localized meningitis, that has not broken through the meninges, but in which the reaction is so intense that an exudate of leucocytes occurs. This is comparable to an ulcer of the cornea, due to a pneumococcus infection in which the organism can be demonstrated in the cornea, but the hypopyon or pus in the anterior chamber is always sterile—essentially a provocative leucocytosis. Again an intense protein reaction will give us a cloudy fluid that clears up. Essentially the organisms have not broken through the meninges. Again meningismus and serous meningitis belong to this type.

*Treatment:* The fact that so many kinds of treatment have been suggested and that the problem has been approached from so many view points, and especially that the death rate is so high, simply indicates that the treatment is still "sub judice." Practically every

method of treatment has been used. The problem has been approached from two main avenues. One is based upon surgery and drainage, such as repeated lumbar punctures, drainage of the cysterna, etc. The second has been the use of antisera and drugs into the spinal fluid, etc. The third has been a combination of the above two. Doctor Kolmer's brilliant work reported tonight, has been of the third class. The writer has tried all three and no real progress can be reported in any series of cases. Eight of the cases in the series got well under different methods. The same methods failed in the vast majority of cases.

*Concluding Thoughts:* With the brilliant results obtained in the treatment of the epidemic form, with a specific antisera, it would seem to the writer, that the greatest hope will be the development of antisera. At present serology has not advanced to this point. We have used surgery, antisera, drugs, leococytic extract, etc., with no convincing results.

Two points are firmly fixed in our minds. The first is that the pathway of infection is very important, and should be studied more extensively to get localized symptoms as to the location, as in petrositis and localizations through the labyrinth, or around the saccus endolymphaticus, etc. The second is that proper surgery in these cases will prevent a localized process from becoming generalized. Once it is generalized according to the above observations, it is an almost hopeless proposition. It is essentially a problem that has not yet been solved.

162 East 46th Street.

# SEPTIC MENINGITIS OF RHINO-LARYNGOLOGICAL ORIGIN.*

DR. LEE M. HURD, New York.

*Etiology and Symptoms:* It is the commonest of all intracranial complications of nasal origin. It occurs as a primary complication, and as the terminal, lethal condition in other intracranial complications, such as extradural abscess, brain abscess, septic thrombophlebitis of the venous cranial sinuses. It is very much less common than septic meningitis of otitic origin.

*Actual cause* is entrance into, and spread within, the leptomeninges of septic organisms, streptococci, pneumococci, and staphylococci, rarely other organisms.

*Exciting causes* are: *a.* Virulent infection, as with influenza, measles, scarlet fever, diphtheria, erysipelas, furuncle of face or nose, and septic infection of the pharynx, tonsils and jaws. *b.* Trauma, especially operations involving the "danger area," of the nose, olfactory cleft; meningitis has followed septum operation, middle turbinectomy, or cauterization of middle turbinate, simple polyp removal, and any sinus operation, fracture involving sinus or nasal cerebral wall. The factors at work are: *1.* Lighting up of virulent infection in a latent sinusitis; *2.* incomplete operation, as failure of drainage of the ethmoids when the frontal is opened, or sphenoid when the ethmoids are opened; *3.* instrumental perforation of the nasal roof or cranial wall of a sinus, with tear of the dura; *4.* prolonged nasal packing in the presence of infection.

*c.* Rhinorrhea, fracture of the skull involving the nose or sinuses may produce immediate infection of the meninges or cause a passage to be formed between the subarachnoid space and the nose. This is due to an extension of the epithelial cells of the nasal mucosa to the arachnoid leaving a permanent passage, resulting in a rhinorrhea, and a subsequent infection in the nose will cause meningitis.

*d.* Postoperative osteomyelitis, especially after opening of diploe and elevation of the periosteum in the removal of the anterior wall of the frontal sinus.

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*c*. Complete blocking of drainage by edema, polyps, etc., of the frontal nasal duct, or of the ethmoid or the sphenoid by edema of the middle turbinate, especially in the narrow noses of children.

*Pathways of Infection of Meninges: a*. Lymphatic connections. Lymph channels connect the nose and the spaces of the pia mater directly *1*. along the sheaths of the olfactory nerve fibres; *2*. from a submucous network in the uppermost parts of the nasal cavities, which drains directly into the spaces of the pia mater. Some of the cases of hyperacute meningitis seen in influenza have been shown to be due to streptococci entering the meninges via these lymphatics directly from the olfactory area of the nose. Other very acute postoperative meningitides are due to too close an approach to the danger area during the operation, especially if latent infection is present.

*b*. Pre-existing bony defects in the cranial walls of the sinuses. Such defects have been shown to occur in the posterior wall of the frontal, in the roof of the ethmoids or in the cribriform plate, and in the roof and lateral wall of the sphenoid. They are rare.

*c*. Perforation of the cranial wall of a sinus by a process of localized necrosis. This has been observed fairly often in the posterior wall of the frontal and in the cranial walls of the sphenoid sinuses.

*d*. The veins; *1*. perforating venules run directly between the mucosa of the frontal sinus and the veins of the dura mater over the frontal lobe, and from the mucosa of the sphenoid sinus to the cavernous sinuses. Infection may thus pass directly to the meninges or the cavernous sinus and thence to the meninges. *2*. Connecting venules run from the posterior ophthalmic vein, which receives the anterior and posterior ethmoid veins from the nose, directly to venules of the dura mater, and into the superior longitudinal sinus. *3*. The main venous drainage of the nasal chamber and sinuses is backward by the sphenopalatine veins into the pterygomaxillary venous plexus, which communicates with the cavernous sinus. *4*. Spreading septic thrombophlebitis of the facial, angular, superior ophthalmic veins and hence of the cavernous sinus may follow furuncle of the nasal vestibule, upper lip or cheek; it commonly ends in septic meningitis.

Cavernous sinus thrombosis with rapidly following fulminating meningitis not infrequently follows trivial staphylococcus infection of the face, such as small furuncles of this area, acute tonsil-

litis, peritonsillar abscess, dental infection, acute or exacerbation of chronic infection in the sphenoid sinus. There is usually a history of trauma to the infection, such as squeezing the furuncle, extraction of tooth, incising abscess in throat, or recent operation on the sphenoid. It reaches the cavernous sinus by direct thrombophlebitis through veins from the infected area. Symptoms, in brief, are swelling of the eyelid, chemosis, high sustained temperature, protrusion of eye, paralysis of the third, fourth and sixth nerves, with the opposite eye soon showing similar symptoms, coma, plus the signs of a fulminating meningitis.

*e*. Osteomyelitis, of the cranium spontaneously, or after operation, very often leads to meningitis. May be slow spread through diploe, or more rapid through the veins of Breschet.

*f*. Orbital suppuration which may itself be secondary to sinus infection. Infection reaches the meninges: *1*. by perforation of the bony orbital wall into the meninges; *2*. through the optic foramen, superior orbital fissure or the sheaths of the nerves entering the orbit; *3*. by a septic thrombophlebitis of the ophthalmic veins.

*g*. Suppuration in the sphenopalatine fossa, secondary to thrombophlebitis of the pterygomaxillary venous plexus, or by rupture into it of suppuration in maxillary sinus or sphenoid sinus. Suppuration reaches the meninges via the foramen rotundum or the superior orbital fissure.

### SYMPTOMS AND SIGNS.

Onset usually sudden, may be slower when process becomes generalized from focus as extradural abscess, or cerebral abscess.

Marked malaise. Severe increasing general headache.

Vomiting, either toxic, or due to intracranial pressure is usually projectile, and there may be no nausea.

Continuous high fever, 104°-105°.

Irregularities in respiration, sometimes Cheyne-Stokes.

Pulse fast, first full, later thready, finally irregular.

Irritability, photophobia, increasing stupor, coma. Localized twitchings, general convulsions, delirium.

Paralyses of ocular muscles, optic neuritis, facial musculature weakness.

Kernig's sign positive, stiffness of posterior neck muscles, later retraction of head, tenderness of muscles. Tache cerebrale, due to deranged vasomotors in skin.

*Lumbar puncture* shows an increase in the amount of fluid, increase in the pressure, increase in foam on shaking fluid in a test tube, increase in the cell count. If they are lymphocytes the prognosis is better than if they are polynuclears. The latter cause the fluid to be turbid to actually purulent, with pellicle or sediment on standing. Increase in the proteins, decrease in the chlorid, compared with the blood chlorid, and sugar tends to disappear, and usually the organisms in smears or cultures are found.

*Diagnosis* is made on the clinical picture, neurologic findings, and proven by the results of lumbar puncture.

*Differential diagnosis* from brain abscess by slower course, slow pulse, normal or slight rise in temperature, Kernig's sign usually is absent. Lumbar puncture may be normal, or show increased pressure. Lymphocytes later decreasing, no organisms.

*Prognosis* is extremely bad, worse by far than otitic meningitis, as few cases of recovery have been reported.

39 E. 50th Street.

# NEWER METHODS FOR THE PROPHYLAXIS AND TREATMENT OF MENINGITIS WITH SPECIAL REFERENCE TO STREPTOCOCCUS AND PNEUMOCOCCUS MENINGITIS.*‡

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While streptococcus and pneumococcus meningitis secondary to otitis media, mastoiditis and sinusitis or accompanying these infections are fortunately uncommon, yet their dreadful mortality renders them among the most mortal of diseases and properly commands great interest and continued efforts towards solving the problem of their prophylaxis and treatment. And despite the splendid progress made in the treatment of epidemic meningococcus meningitis during the past twenty years with antimeningococcus serum, the mortality of this form is still sufficiently high to merit further investigations in prophylaxis and treatment.

My own efforts have extended over a period of ten years and have been concerned not only with the serum, chemotherapeutic and drainage treatment of meningitis experimentally produced in the lower animals with highly virulent pneumococci and streptococci, but likewise with the clinical trial of various methods offering some encouraging results in the treatment of the experimentally produced infections. It is my purpose to herewith briefly summarize the results of this laboratory and clinical experience as well as more recent investigations with Dr. K. W. Amano upon the serum and vaccine prophylaxis of experimental streptococcus and pneumococcus meningitis, and to discuss some of the more important problems in the diagnosis and treatment of these dreadful infections occurring in human beings.

## ROUTE OF INFECTION IN RELATION TO PROPHYLAXIS AND TREATMENT.

In my experience the majority of cases of streptococcus and pneumococcus meningitis have been secondary to infection of the ears, mastoid or nasal accessory sinuses with extension to the meninges. It is stated that hematogenous infections may occur, as is now com-

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monly believed to be the route of infection in meningococcus meningitis; that is to say, that streptococci or pneumococci may enter the blood presumably from the postnasal area and localize in the meninges with or without coincident otitis media, but I have never seen a case in which the evidence of this mechanism was thoroughly established. In other words I believe that the majority of instances of streptococcus and pneumococcus meningitis present important surgical problems referable not only to drainage of cerebrospinal fluid but likewise to the best possible drainage of the primary foci and their thorough surgical removal whenever possible. But not infrequently the impossibility of adequately draining or totally extirpating the primary foci greatly complicates the problem of treatment because of continuous reinfection of the meninges from these areas.

But because of the frequency with which streptococcus and pneumococcus meningitis are secondary to infections of the ears, mastoid and sinuses, I have considered the possibility of prevention by the administration of immune sera immediately after operation or by active immunization with autogenous vaccines when there is sufficient time and opportunity for this preoperative preparation. And the results of experiments along these lines by Dr. Amano and myself, conducted during the past year by reason of the aid granted by the McCarthy fund, have yielded some hopeful results to which further reference will be later made.

It is true that signs and symptoms of meningitis may develop during the course of lobar pneumonia, but it would appear that the majority of these are cases of acute meningeal congestion or meningismus (so-called "serous meningitis") with sterile spinal fluids and in which recovery is the rule. But even if pneumococci and pus are found in the cerebrospinal fluid, the problem of treatment is much simpler in the absence of primary infection in the ears, mastoid or sinuses and the prognosis very much better. It is my impression that the majority of recoveries from pneumococcus meningitis recorded in the literature are of this type and I have recorded instances of recovery following the intracarotid injection of antipneumococcus sera.

It would also appear that streptococcus meningitis may occasionally develop during the course of scarlet fever or other types of streptococcus septicemia without discoverable primary foci in the ears, mastoid or sinuses and the prognosis is likewise much more favorable under these conditions with the recorded recovery of some cases following intraspinal or other types of therapy, but the fact remains that the great majority of cases of streptococcus meningitis are

secondary to primary infections of the ears, mastoid or sinuses in which surgeons must continue to bear a large share of responsibility in treatment because of the importance of extirpating or adequately draining the primary focus or foci which are so likely to be the source of continuous infection of the meninges.

And personally I have as yet never seen a case of real staphylococcus meningitis that was not secondary to primary infection of the ears, mastoid or sinuses, the result of injury or due to infection incident to spinal puncture. Owing to the fact that staphylococci are much less aggressive than pneumococci and streptococci this type of meningitis however, tends to remain localized with a much better prognosis.

### THE PROBLEM OF DIAGNOSIS IN RELATION TO TREATMENT WITH SPECIAL REFERENCE TO SPINAL FLUID EXAMINATIONS.

Nothing can cause more apprehension than signs and symptoms of meningitis following operations upon the ears, mastoid or nasal accessory sinuses with special reference to the ethmoid and sphenoid cells.

As a general rule the early signs and symptoms are indicative of a localized meningitis and usually present no difficulties in diagnosis. But the spinal fluid secured by lumbar puncture may be clear and sterile and lead to the diagnosis of localized "meningismus." And it is very easy to be misled under these conditions and to hope that the meningeal involvement will be but temporary and remain localized with an excellent prognosis.

But in my experience "meningismus" and localized meningitis have usually progressed to diffuse meningitis and death. But one never fails to hope that the meningeal involvement will remain localized and it is indeed very difficult under these conditions to decide upon a radical plan of treatment involving as it may the possibility of further surgery and the extraordinary measures to be shortly described. When the signs and symptoms of diffuse meningitis are present along with evidence indicating that the patient is doomed it does not require any great degree of nerve to try anything in the desperate hope of successfully combating a hopeless situation but during the first day or two, when the patient does not appear to be very sick, it is indeed difficult to decide upon a radical plan of treatment. But in my experience it is necessary to reach this decision as early as possible and attempt the more radical treatment because the great majority of cases go on to diffuse meningitis and a hopeless prognosis. In so far as the treatment of experimental streptococcus and pneumococcus meningitis are concerned, it has been impossible for me to save any

animal when the infection has had more than a forty-eight hour start with diffuse purulent meningitis and I believe it is well for the oto-rhinologist to realize that if anything worth while is to be attempted in treatment, it is necessary to institute radical measures within the first twenty-four to forty-eight hours after the onset of signs and symptoms of meningitis even though these are indicative of a localized infection or indeed of a "localized meningismus," since both are so likely to progress to diffuse meningitis.

So far I have never seen any harm following spinal puncture and the slow withdrawal of about 5 c.c. of cerebrospinal fluid for diagnostic purposes. When the early signs and symptoms are indicative of a strictly localized meningitis, one naturally wonders if the withdrawal of spinal fluid will favor dissemination of the infection and diagnosis is not infrequently delayed because the physician hesitates taking the chance. I usually measure the pressure with a mercury manometer, as this information alone is of diagnostic value, and withdraw 3 to 5 c.c. of fluid (ample for all necessary tests) very slowly and with very gradual reduction of pressure. Under these conditions I have never seen any evidences of ill effects.

Nor have I any fear of favoring the localization of streptococci in the meninges from the blood in cases of septicemia by spinal puncture conducted in this way. It may be that the sudden lowering of cerebrospinal fluid pressure by the withdrawal of large amounts of fluid with a large needle may favor the spread of a localized meningeal infection or result in the localization of streptococci from the blood, but with the slow removal of small amounts of fluid neither of these accidents have occurred in my experience.

Indeed, I have frequently given dogs and rabbits enormous doses of hemolytic streptococci and Type I pneumococci intravenously and have conducted spinal and cisternal punctures with the withdrawal of as much as 0.5 to 1 c.c. of cerebrospinal fluid per kilogram of weight (corresponding to 30 to 60 c.c. for an adult) immediately and at varying intervals thereafter without ever succeeding in producing meningitis by localization of the organisms in the meninges, even though the organisms were secured by blood cultures for a period of hours after inoculation in the dogs, while the rabbits succumbed to the septicemias within 72 hours with positive heart blood cultures.

I believe, therefore, that spinal puncture and the withdrawal of small amounts of cerebrospinal fluid for diagnostic purposes can be made a perfectly safe procedure in the early diagnosis of meningitis and I plead for early examinations because of the frequency with

which considerable aid is given in diagnosis and especially because early diagnosis and the institution of prompt treatment offer some hope of success while undue delay, with the development of diffuse septic meningitis, may result in a well-nigh hopeless situation.

In early localized meningeal congestion (localized "meningismus") secondary to otitis, mastoiditis or sinusitis, the cerebrospinal fluid is clear and normal in appearance, with a total cell count of less than 100 per c.m.m. and with no increase of protein or reduction of sugar along with the absence of organisms in smears and cultures. The pressure may or may not be slightly increased and diagnosis is largely based upon clinical signs and symptoms, associated with these essentially negative findings. Eagleton has shown, however, that in some cases the cerebrospinal fluid obtained from the site of invasion may give positive results and especially positive cultures, and it is a prevailing opinion that cases of this so-called "meningismus" are to be regarded as potentially septic.

In localized meningitis, and I believe that the majority, if not all cases, of meningitis associated with otitis, mastoiditis and sinusitis are primarily localized infections, the fluid obtained within a few hours after the onset of signs and symptoms may show the same characteristics but usually contains more cells, a slight increase of protein, a normal or slightly decreased amount of sugar and may or may not yield a positive culture, although organisms are not usually found in direct smears. The fluid may be clear but is usually opalescent and may be distinctly cloudy, owing to the presence of hundreds or several thousands of polymorphonuclear pus cells per c.m.m. of fluid. Indeed, the absence of organisms in direct smears may be all that is lacking in the characteristic spinal fluid changes of septic meningitis, but this should not be allowed to give a false sense of security as it is quite usual for subsequent fluids, drawn 12 to 24 hours later, to show streptococci or pneumococci in small numbers, followed later by larger numbers as the infection spreads.

Here again there is a great temptation to wait further developments and especially if the patient does not appear to be very sick and if the surgeon believes that he has already extirpated as much infected bone as necessary, with adequate drainage. But the frequency with which the infection continues to spread in the following few days, with the development of fatal meningitis, has given me the conviction that it is better practice to immediately institute drainage and other remedial measures while in the treatment of diffuse septic meningitis, characterized by definitely increased spinal fluid pressure and cerebrospinal fluids which are frankly purulent with

several thousands of pus cells per c.m.m. along with greatly increased protein, an absence of sugar with the presence of organisms in direct smears (even though but few) and positive cultures, a radical combination of surgical drainage and serochemotherapeutic procedures should be instituted as promptly as possible if it is decided to give the patient some chances of recovery, even though it is realized at the outset that under these circumstances recovery is indeed doubtful.

### THE SURGICAL REMOVAL AND DRAINAGE OF PRIMARY FOCI OF INFECTION IN RELATION TO THE TREATMENT OF SEPTIC MENINGITIS.

If we had available a chemotherapeutic agent capable upon the intravenous or subthecal injection of safe doses of specifically destroying pneumococci and streptococci in the tissues in a manner analogous to the destruction of *spirochacta pallida* by arsphenamin and its congeners, the ideal in the treatment of septic meningitis would be attained. But while we are in no sense anywhere near this goal, although I believe it will be ultimately reached if enough money and capable workers are available for the extensive and expensive investigations required, surgical drainage must play an important rôle in the treatment of septic meningitis in conjunction with serochemotherapeutic measures.

I agree with Hempstead[1] that upon the earliest signs of meningitis, when the clinical signs and spinal fluid findings indicate the presence of only a localized "meningismus" or meningitis, that there should be a complete exenteration of the cells in cases of acute mastoiditis, a radical operation in cases of chronic suppurative otitis media and the best possible drainage in sinusitis; if the primary focus has been the labyrinth, thorough drainage should be established through this area. And in all cases in which the dura is exposed there should be a wide incision at the site of operation for local drainage of cerebrospinal fluid. If the dura looks good and healthy I can well understand the natural hesitation of incising it for fear of opening up an avenue of infection, but I have not yet seen this occur and believe that the frequency with which fatal meningitis follows anyhow if it is not incised justifies the procedure.

### SPINAL FLUID DRAINAGE IN RELATION TO THE TREATMENT OF SEPTIC MENINGITIS.

Furthermore, there is a difference of opinion upon the advisability of establishing spinal fluid drainage when the meningitis appears to be localized for fear that a lowering of spinal fluid pressure may favor a dissemination of the meningeal infection.

So far I have not been able to secure reliable data on this point by experiments on monkeys, dogs and rabbits; but Bowers[2], Weaver[3], Huenekens and Stoesser[4], Peterman[5], Hempstead[6] and others have reported cures from repeated spinal punctures with drainage of fluid, and I personally believe that it is the proper thing to do at frequent intervals; that is to say, to make a spinal puncture, measure the fluid pressure and slowly drain off enough fluid each time to reduce the pressure to about 2 to 4 m.m. of mercury. If these procedures are accompanied by clinical improvement, along with a diminution of spinal fluid changes, a conservative policy of this kind may be all that is required.

But if the the infection is progressive, as indicated by an aggravation of the signs and symptoms along with progressive cerebrospinal fluid changes after one or two days of this conservative treatment, I believe that it is necessary to consider a more radical plan, as is likewise true in fulminating cases of diffuse meningitis with pronounced spinal fluid changes, because a cleaning out of the primary focus with but one spinal drainage a day has proven unsuccessful in the great majority of cases.

Furthermore, it would appear advisable to institute a more radical treatment without undue delay in order to obtain the possible benefits before the exudates become plastic. I well realize, however, how difficult it is to reach this decision at the bedside when the patient is fully conscious and in good general condition, but there should be a wider recognition of the fact that a spreading septic meningitis with purulent spinal fluids showing the presence of streptococci or pneumococci constitutes one of the most mortal of diseases in which only a radical plan of treatment, instituted promptly and before the patient becomes moribund, may afford the only chance of recovery under conditions where there is nothing to lose and possibly something to be gained by a bolder policy.

From the surgical standpoint this may consist of spinal drainages every six to eight hours while the intake of fluids is increased. Or laminectomy may be performed and continuous drainage instituted, as reported by Hill[7], Rainey and Alford[8], and more recently advocated by Spurling[9]. The latter, who reports eight cases with five recoveries, then forces the intake of fluids to 4 or 5 liters a day, believing that this procedure affords continuous drainage of inflammatory exudates with reduction of cerebrospinal fluid pressure and dilution of the toxins and other products of the inflammation.

Owing to technical difficulties I have not been successful in establishing continuous spinal drainage in dogs and monkeys with experi-

mental pneumococcus and streptococcus meningitis, and in two cases of severe hemolytic streptococcus meningitis of adults this plan of treatment failed, but I have observed the recovery of dogs and rabbits with Type I pneumococcus meningitis following continuous drainage through trephine openings of the skull with incision of the dura made within 24 to 48 hours after the onset of meningitis following intracisternal inoculation with virulent cultures and before plastic exudates had formed.

In 1912 Haynes[10] described an operation for continuous drainage of the cisterna magna, and in 1924 Dandy[11] reported the recovery of three out of a series of four cases of meningitis due to staphylococci and streptococci by this procedure; Sharpe[12] has expressed himself in favor of subtemporal drainage, but it would appear that these procedures have failed to fulfill original expectations and are no longer practiced; indeed, as high an authority as Eagleton[13] states that subarachnoid irrigation is impossible for mechanical reasons and drainage of the system fallacious. However, it is practically certain that continuous cerebrospinal fluid drainage alone is not to be depended upon for the treatment of septic meningitis, although worthy of serious consideration in combination with serochemotherapeutic measures. Personally, I believe that spinal punctures with thorough drainage of cerebrospinal fluid every six to eight hours may meet the requirements, although laminectomy with continuous spinal drainage is worthy of further trial in view of the results reported by Spurling.

### VENTRICULAR-CISTERNAL-SPINAL LAVAGE IN THE TREATMENT OF SEPTIC MENINGITIS.

Five years ago, we[14] found that we were able to save an encouraging percentage of dogs with Type I pneumococcus and hemolytic streptococcus meningitis by lavage with 20 to 40 c.c. of warm saline or Ringer's solution from a lateral ventricle to the cisterna magna with or without lavage of the spinal subarachnoid space to the lumbar region. The method required a trephine for puncture of a ventricle along with the passage of needles into the cisterna and lumbar region of the cord but the whole was quickly and readily conducted. Following the lavage we sometimes improved the results by injecting pneumococcus antibody solution, antipneumococcus serum or antistreptococcus serum into the ventricle or great cistern and thought that the results offered an encouraging advance in treatment. Since the injection of the immune sera alone failed to save any of the animals we naturally laid most emphasis upon the lavage and as a general rule one treatment sufficed.

A little later we were able to try the method on two very severe and moribund cases of pneumoccus meningitis but without recoveries. Autopsies revealed that while the ventricles, great cistern and spinal subarachnoid space were remarkably free of pus, yet the cerebral subarachnoid space was filled with plastic exudates (as was likewise found in some of the dogs) and indicating that the ventricular-cisternal-spinal lavage and medication were insufficient for reaching the cerebral subarachnoid infection.

### CEREBRAL-CISTERNAL-SPINAL LAVAGE AND MEDICATION IN THE TREATMENT OF SEPTIC MENINGITIS.

I still believe, however, that if it were possible to treat human beings as early as one does dogs with the experimentally produced infections and before the exudates became plastic, ventricular-cisternal-spinal lavage with physiologic solution of sodium chlorid, followed by the injection of antibody solution or antistreptococcus serum, according to the nature of the infection, would probably prove of value; however, difficulties and delays in diagnosis, the hope that the meningitis will remain localized and the natural hesitation to resort to bilateral trephining for ventricular irrigation and treatment have resulted in inevitable delays.

Our attempts to establish continuous drainage of the cisterna magna and spinal meninges have not encountered any success in the treatment of dogs. While we were engaged in experiments on cerebral subarachnoid lavage in addition to cisternal and spinal lavage and medication, Stewart published his paper[15] on the results of treatment for experimental Type I pneumococcus infections of dogs by this quadruple method, consisting of the injection of serum and ethyl-hydrocuprein hydrochlorid (optochin) mixtures into the cerebral subarachnoid space (both sides), cisterna magna and lumbar region of the cord. Dr. Stewart employed a mixture of 15 c.c. of Type I antipneumococcus serum with 0.75 c.c. of a 1 per cent solution of ethyl-hydrocuprein hydrochlorid and found that when the drainage was excellent and that the drug had no effect on the respiratory center, progressive sterilization of the meninges with some recoveries were recorded.

We have had similar encouraging results[16] in the case of dogs infected by the intracisternal injection of 0.1 c.c. of from six to 18 hours hormone broth cultures of a virulent Type I strain of pneumococcus. Within 24 hours severe diffuse fibrinopurulent meningitis has occurred, with cloudy spinal fluids teeming with pneumococci, and has invariably proved fatal in from four to seven days in the case of the dogs used as controls.

From 24 to 48 hours after injection and when symptoms and signs of meningitis were well marked, with purulent cisternal and spinal fluids, the dogs were placed under ether anesthesia, and the following treatment given: *a*. Double frontal trephines. *b*. Introduction of needles into the lumbar subarachnoid space and cisterna magna. *c*. Lavage with warm physiologic solution of sodium chlorid from lumbar to cisternal needles until the solution returned clear (usually from 10 to 25 c.c.). *d*. The injection of 2 c.c. of antibody and ethyl-hydrocuprein hydrochlorid mixture* into each cerebral subdural space, followed by the intracisternal injection of 3 c.c. and the intraspinal injection of from 7 to 10 c.c. of the same mixture and withdrawal of the needles.

Immediate recoveries generally occurred, although an occasional animal died from respiratory failure ascribed to the severe meningeal infection. As a general rule, treatment was repeated two or three times at approximately 25-hour intervals. All of the dogs used as controls succumbed in from four to seven days from severe fibrinopurulent diffuse leptomeningitis.

Many of the animals recovered permanently (approximately 30 per cent), likewise a smaller number treated in the same manner with similar doses of Type I antipneumococcus serum and ethyl-hydrocuprein hydrochlorid mixture, as well as some treated with antibody solution and serum alone without the addition of ethyl-hydrocuprein hydrochlorid.

However, much to our surprise, some animals treated in exactly the same manner with physiologic solution of sodium chlorid alone recovered without the subsequent reinfections described by Dr. Stewart and ascribed to foci of infection which escaped disinfection. Dogs treated with ethyl-hydrocuprein hydrochlorid alone in physiologic solution of sodium chlorid did not give an appreciably higher percentage of recoveries. For this reason I believe that the drainage and lavage alone with the mechanical removal of most of the infection is of considerable importance, although there can be no doubt that the injection of the antibody and ethyl-hydrocuprein hydrochlorid and serum and ethyl-hydrocuprein hydrochlorid mixtures gave a higher percentage of permanent recoveries, which in my opinion was due in larger part to the effects of the antibody solution or serum than to the ethyl-hydrocuprein hydrochlorid.

In numerous instances, however, it was difficult and indeed impossible to irrigate thoroughly the cerebral subarachnoid spaces by

*A mixture of 30 c.c. of pneumococcus antibody solution (Mulford) and 1.5 c.c. of a 1 per cent solution of ethyl-hydrocuprein hydrochlorid (ethyl-hydrocuprein hydrochlorid).

this method even when the treatment was instituted 24 hours after the onset of infection; necropsies on the dogs treated which succumbed to the diseases invariably revealed areas of fibrinoplastic meningitis evidently untouched by the lavage and medication. Furthermore, I have not been able so far to secure favorable results in the treatment for meningitis in human beings by cerebral-cisternal-spinal lavage and medication. I hasten to add, however, that all five cases in which the method has been tried were late cases and two of the patients were moribund when the treatment was instituted. In every case it was impossible to introduce satisfactorily the antibody or serum ethyl-hydrocuprein hydrochlorid mixtures into the frontal cerebral subarachnoid spaces because of obliteration, due to plastic exudates. In each instance most of the mixture returned through the meningeal openings so that the surgeons and I were doubtful if any diffused through the cerebral subarachnoid space; at least, it was certain that none reached the cisternas. In the dogs, however, there was no doubt of the success of the injections into the subarachnoid space, evidently because the injections were given before plastic exudates had formed to sufficient degree to cause obliteration of the subarachnoid space. In one of the five cases in human beings a vein was unfortunately punctured, which caused profuse bleeding and the formation of a large subarachnoid hematoma.

I am certain, therefore, that the results of this method of cerebral cisternal spinal lavage and medication observed in meningitis experimentally produced in dogs cannot be duplicated in human beings when the treatment is delayed to the point at which the subarachnoid fluid is extremely purulent with plastic exudates.

Unfortunately, the early production of fibrinous exudates is characteristic of pneumococcus infections; they are less rapidly produced by streptococci. However, if the treatment were instituted earlier in the course of the disease, within from 24 to 48 hours after the onset of signs and symptoms and when the spinal fluid was only slightly cloudy instead of frankly purulent, the results might be more encouraging. Certainly, the method is severe, including as it does bilateral trephining; but, on the other hand, diffuse pneumococcus and streptococcus meningitis are highly fatal diseases with mortality rates of almost 100 per cent and thereby justifying any measure offering even a hope of success. I am confident that the operation itself is without particular danger, providing facilities are at hand for rapid trephining and the removal of large enough buttons of bone to permit injections into the subarachnoid space without the puncture of meningeal veins. Ethyl-hydrocuprein hydrochlorid

in the amount employed (1 c.c. of 1:100 solution to 25 c.c. of antibody solution or serum), which was slightly less than that used in the treatment for dogs, is apparently well borne without evidence of effects on the respiratory center.

THE INTRACAROTID METHOD OF TREATMENT FOR SEPTIC MENINGITIS.

But the great difficulty and indeed the impossibility of bringing medicaments in contact with all of the meninges infected in severe septic meningitis by cerebral-cisternal-spinal lavage and medication, especially when the subarachnoid exudates have become plastic, has led me to try the treatment of pneumococcus and streptococcus meningitis produced in dogs by injections of immune sera and chemical agents into both common carotid arteries. Injections of dyes have shown an intense and widely distributed concentration of inoculum in the cerebral and basilar meninges, reaching as far as the upper end of the cord, with the passage of a portion into the cerebrospinal fluid.

Furthermore, the ease and safety with which Hirsch, Myerson, Halloran and others have given intracarotid injections of neoarsphenamin and sulpharsphenamin in the treatment for paresis have shown that this route of medication might be used with safety in human beings if encouragement were found in the treatment for experimentally produced meningitis in dogs.

The results of numerous experiments[17] have shown that injections into the common carotid arteries can be given with practically no bleeding; that the method is both simple and safe; that the toxicity of drugs is no higher per kilogram of weight than by intravenous injection; that intense concentration of the medicament occurs in the meninges, and that encouraging results have been observed in the treatment of severe pneumococcus and streptococcus meningitis in dogs when the bilateral injections were combined with cisternal drainage and intracisternal medication.

Dogs infected by the intracisternal injection of young broth cultures of virulent Type I pneumococci or highly virulent hemolytic streptococci developed symptoms within 24 to 36 hours and succumbed within three to six days with all the classic signs of fulminating meningitis, including the characteristic changes in the cerebrospinal fluid.

But from two to six dogs out of each 10 with pneumococcus meningitis made complete recoveries when the following treatment was instituted within 24 hours after infection and when symptoms of meningitis and spinal fluid changes were first apparent: *a*. cisternal drainage; *b*. the injection into each common carotid artery of

0.5 c.c. per k.gm. of weight of a mixture of polyvalent antipneumococcus serum or Huntoon's antibody solution and a 1:100 solution of optochin (ethyl-hydrocuprein) hydrochlorid* followed by *c.* the intracisternal injection of 0.5 c.c. of the mixture.

During the past two years, however, similar experiments conducted with the assistance of Dr. K. W. Amano and Miss Anna Rule, employing Felton's concentrated antipneumococcus serum, kindly furnished by manufacturers, and a similar concentrated serum prepared by the Massachusetts Department of Health and kindly furnished by Dr. Benjamin White, have yielded somewhat better results, probably because they contained more Type I antibody than the serums previously employed.

In experimental streptococcus meningitis the results of this plan of treatment were much less encouraging, probably because of a lack of sufficient antibody in ordinary polyvalent antistreptococcus serum. Somewhat better results were observed, however, with the intracarotid and intracisternal injections of scarlet fever antitoxins, probably because of the higher antibody content for the hemolytic strain of streptococcus employed in our experiments.

Since a large number of experiments at least proved that the method of treatment was entirely safe and apparently based on a sound theoretical basis, in that antibacterial agents were brought in high concentration to the infected tissues even though for but a brief period of time, the method has been employed in the treatment of pneumococcus, streptococcus, staphylococcus and meningococcus meningitis of human beings during the past three years. At the outset, however, it was hardly hoped that exactly similar results could be obtained in pneumococcus and streptococcus meningitis because the inevitable delays in diagnosis make it impossible to start the treatment at the outset of the disease or before plastic exudates are formed. Furthermore, in the experimental disease of dogs, we were not dealing with primary foci of suppuration in the nasal accessory sinuses or mastoid cells, as is so commonly the case in septic meningitis of human beings, and resulting in continuous infection or reinfection of the meninges.

I believe that pneumococcus meningitis is more amenable to this method of treatment than streptococcus meningitis because of the more potent antipneumococcus sera available, along with the fact that ethyl-hydrocuprein hydrochlorid may be added to the serum for intracarotid and intracisternal or intraspinal injection. Of four cases treated during the past two years, one recovered[18].

*One c.c. of a sterile 1:100 solution added to each 20 c.c. of serum or antibody solution.

Unfortunately, however, septic meningitis of otitic origin is much more frequently due to streptococci for which less potent sera and chemotherapeutic agents are available. Of 10 cases treated during the past three years, two recovered under intracarotid injections of mixtures of 10 c.c. of Pregl's solution of iodin and 10 c.c. of a concentrated polyvalent antistreptococcus serum. Dr. Albert S. Crawford, of the Henry Ford Hospital of Detroit, has also reported one recovery in a series of 10 cases following intracarotid injections of Pregl's solution of iodin.

Dr. Crawford[19] also records two recoveries in a series of four cases of severe staphylococcus meningitis following intracarotid injections of Pregl's solution of iodin and also two recoveries in a series of four cases of very severe meningococcus meningitis following intracarotid injections of mixtures of 10 c.c. of Pregl's solution of iodin and 15 c.c. of antimeningococcus serum. I have also recorded the recovery of one case of fulminating meningococcus meningitis from intracarotid and intracisternal injections of the serum alone, and all of these had previously received intraspinal injections of serum without therapeutic effects.

The clinical and spinal fluid improvement following intracarotid therapy is usually very marked and in several cases of pneumococcus and streptococcus meningitis has reached the point where recovery was expected, but the immune sera and chemical agents available at present were unable to effect complete sterilization since death ensued some weeks later from cerebral abscesses. It would appear that the method represents an improvement but that final success must await the production of more therapeutically potent antibacterial agents.

### THE FUTILITY OF INTRAVENOUS AND INTRASPINAL TREATMENT OF SEPTIC MENINGITIS.

It is true that these methods are radical, and even severe, but it would appear worthless and hopeless to rely upon the less radical and ordinary intravenous and intraspinal methods.

I have tried[20] intravenous injections of ethyl-hydrocuprein hydrochlorid, mercurochrome, metaphen, gentian violet, the acridin dyes, acriflavin base and rivanol and, more recently, the complex Pregl's iodin solution, without the slightest effect; also intramuscular and intravenous injections of pneumococcus antibody solution and serum and polyvalent antistreptococcus serum. It may be that these substances are helpful in combating toxemia and bacteremia but are without demonstrable effect on the course and outcome of experimental septic meningitis. I have also used all of them excepting rivanol in the treatment of human infections, without success.

I have used ethyl-hydrocuprein hydrochlorid, mercurochrome, metaphen, gentian violet and the acridin dyes, acriflavin and rivanol, intraspinally with similar disappointing results in the treatment of experimentally produced infections; also ethyl-hydrocuprein hydrochlorid and gentian violet in human infections, without results.

So far as the administration of these substances by intravenous and intraspinal injection or by the two routes combined is concerned, I am satisfied that nothing is to be hoped for in the treatment of generalized or diffuse pneumococcus or streptococcus meningitis in either man or dog. This has also been found true of intraspinal injections of ethyl-hydrocuprein antibody solution, ethyl-hydrocuprein antipneumococcus serum mixtures and of Lamar's mixtures of Type I antipneumococcus serum with boric acid and sodium oleate (serum, 4 c.c.; 5 per cent aqueous solution of boric acid, 15 c.c.; 2 per cent aqueous solution of sodium oleate, 1 c.c.).

### THE SERUM PROPHYLAXIS OF PNEUMOCOCCUS AND STREPTOCOCCUS MENINGITIS.

In view of the unsatisfactory results observed in the treatment of septic meningitis of human beings, I have turned during the past year to the question of the possible prevention of pneumococcus and streptococcus meningitis by the administration of antipneumococcus and antistreptococcus sera. Since it is usually possible to determine promptly and before operation the bacteria-producing infections of the sinuses and the middle ear and to include bacteriologic examination at the time of operation on the mastoid cells, the question arises whether or not it is possible and advisable to institute specific serum or chemotherapeutic phophylactic measures before or within a few days after operation in the case of infection caused by pneumococci and streptococci. Chronic infections of the middle ear and sinuses are not infrequently due to staphylococci, *B. Friedlander, B. pyocyaneus* and other organisms, but apparently these organisms are of much lesser importance in relation to the occurrence of diffuse septic meningitis.

My interest in prophylaxis in pneumococcus meningitis dates back to 1920, when Idzumi and myself first showed that intraspinal injection into rabbits of ethyl-hydrocuprein hydrochlorid alone or in combination with antipneumococcus serum had prophylactic effects when it was administered within from four to six hours after infection of the meninges with virulent Type I pneumococci.

The results of extensive experiments performed during the past year with Dr. Amano[21] with the lower animals indicate that it may

be possible to prevent pneumococcus and streptococcus meningitis as a complication secondary to infections of the sinuses, middle ear and mastoid. Our results have indicated that the intravenous and intramuscular administration of newer concentrated antipneumococcus serums to monkeys and dogs after intracisternal inoculations of virulent Type I pneumococcus had some prophylactic value in the prevention of meningitis and that the intravenous and intramuscular administration of those antipneumococcus serums to rabbits after inoculation of virulent Type I pneumococci into the mastoid and the intratympanal and intracisternal areas had definite prophylactic value in the prevention of meningitis and septicemia, although less definite prophylactic results were observed in rabbits inoculated intracisternally with virulent Type II and Type III pneumococci. The simultaneous administration of ethyl-hydrocuprein hydrochlorid by intravenous and intramuscular injection and of ethyl-hydrocuprein base internally, probably enhanced the prophylactic activity of the antipneumococcus serums.

While in rabbits the intravenous and intramuscular injection of scarlet fever antitoxins had some prophylactic value in the prevention of meningitis and septicemia produced by the intracisternal inoculation of virulent hemolytic strains of streptococci and the results less encouraging yet.

We believe that routine bacteriologic examinations should be made in chronic infections of the sinuses before operation and in mastoiditis at operation, to be followed by the prophylactic measures outlined later under clinical recommendations in cases of unusual severity in which meningitis may occur.

### THE VACCINE PROPHYLAXIS OF PNEUMOCOCCUS AND STREPTOCOCCUS MENINGITIS.

Furthermore, in cases of chronic sinusitis, especially ethmoiditis and sphenoiditis, as well as in some cases of chronic otitis media requiring operative procedures, it is proper to inquire into the possibility of raising immunological resistance by the preliminary administration of autogenous vaccines to the point where some protection may be afforded against meningitis whenever there is sufficient time for such preoperative preparation. This would appear to be particularly indicated when very careful bacteriological examination shows the presence of pneumococci or streptococci, since these are of particular importance from the standpoint of meningitis.

Dr. Amano and I have recently investigated this subject[22] and have found that it is possible to raise the resistance of rabbits to menin-

geal infection with virulent pneumococci and streptococci by preliminary immunization with autogenous vaccines. Especially encouraging results were observed among rabbits vaccinated with Type I pneumococcus and later inoculated intratympanally with the same strain. With Types II and III pneumococci and hemolytic streptococcus the results were less encouraging but we believe that if time permits, careful bacteriological examination of the secretions should be made in chronic sinus and otitic infections, especially ethmoiditis, and autogenous vaccines administered over a period of six to eight weeks to raise immunological resistance preliminary to operation.

### CLINICAL RECOMMENDATIONS.

In conclusion it may be helpful to summarize the results of these clinical and laboratory investigations upon the prophylaxis and treatment of septic meningitis in the form of clinical recommendations or suggestions as follows:

1. *Vaccination:* In selected cases of chronic sinusitis and otitis and especially ethmoiditis, where extensive operative procedures are required, with the possibility of secondary meningitis, it is recommended that very careful bacteriological examinations be made with the preparation of autogenous vaccines containing approximately 1,000,000,000 per c.c. When time permits at least six to eight doses should be given at intervals of four to seven days. For adults the first dose may be 0.2 c.c. by subcutaneous injection and subsequent doses gradually increased until 1 c.c. is being given. Intracutaneous injections of 0.2 c.c. per dose (not increased) may result in bette immunization. Operation to be done about one week after the last dose.

2. *Serum Prophylaxis:* Fortunately, meningitis develops in only a small percentage of cases of infection of the sinuses and mastoids but it is hoped that clinical experience and knowledge have arrived at the point at which certain types of infection are known to be particularly dangerous from the standpoint of meningitis, and that at least some attempt will be made to protect patients from them.

At least, it is usually possible to ascertain before operation the etiologic nature of chronic infections of the sinuses and otitis media, and it is always possible to make smears and cultures of mastoid cells at the time of operation and thereby ascertain the nature of the infection within from 24 to 48 hours thereafter. In this connection it is proper to place emphasis on the necessity of using blood agar or brain hormone dextrose broth (the latter preferred) for making

cultures, as streptococci or pneumococci may fail to develop on plain agar, and this may readily lead to error.

Not infrequently, cultures of pus from infected sinuses and middle ears show the presence of staphylococci in pure culture, and these organisms are very much less likely to result in meningitis, but this conclusion as to the nature of the infection is acceptable only when the cultures have been properly made on either of the aforementioned enriched mediums or on suitable substitutes by an otorhinologist.

If pneumococci are found it is advisable to determine the type, which requires an additional 18 to 24 hours, because this information is of value from the standpoint of dosage of serum and prognosis.

In infections of adults with Type I pneumococcus, 10 c.c. of Felton's concentrated antipneumococcus serum may be injected intramuscularly just before or immediately after operation; a second dose may be given the following day, and a third dose four days later. In infections with pneumococcus Type II and Group IV, the same dosage, or 15 c.c., should be used; in infections with Type III pneumococcus, a dosage of 20 c.c. should be employed.

The same doses may be given to children over 10 years of age; half doses to younger children.

In infections with the pneumococcus, 5 gr. (0.324 gm.) ethylhydrochlorid with milk may be given every five hours for three days after operation in addition to the serum; children require less, according to weight.

In infections with the streptococcus, a therapeutic syringeful of concentrated polyvalent antistreptococcus serum may be given just before or immediately after operation, repeated the following day and again four days later (in unusually severe infections), or an intramuscular injection may be given for the first dose, followed by one or two doses at the same intervals. Children under 10 years may receive about one-half of these amounts.

*3. Treatment of Early Meningitis:* Upon the development of signs and symptoms of meningitis, spinal puncture may be conducted for the slow removal of about 3 to 5 c.c. of fluid. If examination discloses the presence of a localized "meningismus" or meningitis there should be a complete exenteration of the cells in cases of acute mastoiditis and radical operation in cases of chronic suppurative otitis media. If the portal of infection has been the labyrinth, thorough drainage should be established through the labyrinth and in all cases it is advisable to incise the dura at the site of operation. In sinus cases very careful thought should be given to reoperation

for the purpose of affording the best possible drainage. The intake of fluids may be increased and spinal drainage conducted every six to eight hours. Intracarotid and subthecal medication may be omitted.

*4. Treatment of Diffuse Meningitis:* If the above measures fail to check the infection or if the clinical and spinal fluid examinations disclose a spreading type of meningitis more radical measures should be decided upon without delay because there appears to be little or no hope of success in the treatment of advanced cases.

The operative procedures outlined above require first consideration and attention for the purpose of affording the best possible local drainage of the portal of infection.

Laminectomy and continuous spinal drainage should be seriously considered, followed by forcing the intake of fluids up to 4 or 5 liters a day (Spurling).

If the *intracarotid method* is decided upon the treatment may be arranged as follows: For Type I pneumococcus meningitis the injection of 10 c.c. of Felton's concentrated antipneumococcus serum, reinforced with 0.5 c.c. of 1:100 optochin hydrochlorid, into each common carotid artery. Immediately afterward, a cisternal puncture should be made with thorough drainage of fluid and the slow intracisternal injection of from 3 to 5 c.c. of the mixture. Or if cisternal drainage cannot be done, a thorough spinal drainage may be made, followed by the intraspinal injection of from 10 to 20 c.c. of the serum-optochin mixture. Six hours later another spinal drainage may be made, followed by additional drainages every six hours while the intake of fluids is pushed to 4 or 5 liters a day for adults, and children in proportion. This covers the treatment for 24 hours and should be repeated daily for at least two to five or more days, regardless of the clinical improvement of the patient.

In Type II pneumococcus meningitis the same plan of treatment may be followed except that the dose of serum injected into each artery is raised to 15 c.c. and in Type III infections to 20 c.c., since the serum contains much less antibody for these types. I have had no experience with Group IV infections, but since the serum has not been without some benefit in the treatment of Group IV pneumonias, despite the absence of antibody and probably because of nonspecific protein effects, it may be worth trying in the 10 c.c. dosage.

In streptococcus meningitis the same plan of treatment may be followed except that about 10 c.c. of scarlet fever antitoxin or concentrated polyvalent antistreptococcus serum mixed with 10 c.c. of Pregl's solution of iodin are injected intracarotidally and the serum alone intracisternally or intraspinally. It is also necessary to conduct

blood cultures at frequent intervals and especially if lateral sinus thrombosis is suspected or known to be present.

If serum cannot be employed, one may inject 10 c.c. of 0.5 per cent solution of neutral acriflavin or 10 c.c. of concentrated Pregl's solution of iodin into each common carotid artery followed by cisternal or intraspinal injection of these agents, as the results of our animal experiments with them have met with no success. The drainages, however, should be repeated every six hours; this procedure finishes the program for 24 hours and should be repeated daily for at least three to five or more days.

In staphylococcus meningitis, Pregl's solution of iodin may be employed in the same manner as used with success by Dr. Crawford, as previously described, or one may employ neutral acriflavin as described.

Finally, it would appear that, in severe meningococcus meningitis resistant to ordinary intravenous and intraspinal treatment with antimeningococcus serum, the injection of 10 c.c. of serum into each common carotid artery, followed by cisternal drainage and the injection of from 10 to 30 c.c. may be worth while. Here again it would appear advisable to drain at eight-hour intervals and to repeat the serum injections at daily intervals.

Possibly *intracarotid treatment may be combined with continuous spinal drainage by laminectomy and the forced intake of fluids up to 4 or 5 liters a day;* this would appear to be a more hopeful procedure.

The quadruple or *cerebral-cisternal-spinal lavage method* has not proven as hopeful in results, and if tried at all must be instituted as promptly as possible before the inflammatory exudates have become plastic. It may be conducted somewhat as follows for the treatment of *pneumococcus* meningitis:

*a.* Ether anesthesia and bilateral trephining at points well upward toward the longitudinal sinus and well forward in the frontal region to obtain the maximum lavage of the cerebral hemispheres and bathing with Felton's concentrated serum and ethyl-hydrocuprein hydrochlorid mixtures.

*b.* The introduction of needles into the lumbar subarachnoid space and cisterna magna followed by the drainage of fluid.

*c.* The introduction of warm physiologic solution of sodium chlorid into the lumbar region until the return solution from the cistern is practically clear (as determined by collection in a test tube, as inspection of the fluid as it flows from the needle is deceptive). From 25 to 50 c.c. of physiologic solution of sodium chlorid may be required.

*d.* The injection of serum and ethyl-hydrocuprein hydrochlorid mixture* through the lumbar needle until it flows from the cisternal needle, followed by removal of the lumbar needle.

*e.* With the cisternal needle in position, cerebral subdural injection of from 5 to 10 c.c. of mixture on each side.

*f.* The intracisternal injection of about 5 c.c. of mixture, providing there are no respiratory difficulties, followed by removal of the needle.

*g.* As an aid in combating toxemia and septicemia (indicated by positive blood cultures), it may be advisable to inject serum intravenously.

The doses advised are for persons of 12 years and upward; young children may receive less in proportion to age and size.

The outlook for patients with Type I and Type IV pneumococcus infections is doubtless much more hopeful than that for patients with Type II and Type III infections, but the same plan is advised for all.

In *streptococcus meningitis*, the same plan of treatment is advised, although the outlook for success is less than in pneumococcus meningitis, even though the exudates become less rapidly plastic because polyvalent antistreptococcus serum is less efficient than pneumococcus antibody solution and Type I antipneumococcus serum. Furthermore, there is no chemotherapeutic agent for streptococcus infections comparable to ethyl-hydrocuprein hydrochlorid for pneumococcus infections, although I believe that the addition of gentian violet or neutral acriflavin to antistreptococcus serum appreciably enhances its streptococcidal activities. A concentrated polyvalent antistreptococcus serum, therefore, may be employed, and to each 25 c.c. one may add 1 c.c. of a 1:100 solution of gentian violet (Coleman and Bell's) or acriflavin in sterile saline solution.

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# CHRONIC OTORRHEA AND ITS TREATMENT, WITH SPECIAL REFERENCE TO THE USE OF "IODIN DUSTING POWDER" AND "ZINC IONIZATION."*

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Chronic otorrhea, chronic running ear and chronic suppurative otitis media are common synonymous terms interchangeably employed to designate an inflammation of the middle ear, characterized by a purulent discharge over a period of months or years.

The majority of cases of chronic otorrhea arise in childhood following some acute upper respiratory infection or acute exanthema. It is more commonly found among the poor than among the wealthy.

The chief cause of chronic suppurative otitis media is an inefficient care of an acute inflammation and the virulence of certain characteristic micro-organisms, combined with the lowered general resistance of the individual after the acute attack. Chronic suppuration is more likely to follow scarlet fever, measles and epidemic influenza. Tuberculosis, syphilis and diabetes also tend to prolong the suppuration and induce the chronicity. Other important factors are the presence of adenoid vegetation in the nasopharynx, enlarged and infected tonsils, dental caries, deformed nasal septum and turbinates, as well as an infection of the nasal accessory sinuses.

The exciting cause of all suppurative conditions of the middle ear is bacterial invasion[7, 8]. The most common micro-organisms found in the aural discharge are hemolytic and nonhemolytic streptococci, pneumococci, staphylococci albus and aureus, B. mucosus, B. proteus, B. pyocyanus, B. Friedlander, etc.[17] The bacteria that cause the acute condition may be the cause of the chronic, but in the majority of long standing cases, there is a mixed infection, due either to the introduction of new organisms during the subacute attacks or to the invasion of saprophytes from the external auditory meatus. The original infecting organism may have become attenuated or lost its virulence or else the patient has developed an immunity.

The pathological changes of chronic otorrhea depend greatly on the length of time and the extent of the disease. In most cases the lesion is confined to the tympanic cavity. Perforations of the tym-

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panic membrane in various forms are usually seen. The ossicles, especially malleus and incus, may be partially necrosed or totally destroyed[9]. A destruction of the ciliated epithelium and hyperplasia of the mucous membrane in the middle ear often take place. Granulations or polypi may be present, obstructing the canal or blocking the view of the drum head. The epithelium of the external ear may invade the middle ear through the marginal or an attic perforation and may lead to the formation of cholesteatoma, which often causes pressure necrosis of the deeper structure of the middle ear. An intracranial or labyrinthine complication may follow. As a matter of fact, in all long standing cases there is always more or less rarefaction or a sclerotic change of the mastoid process, as well as of the antrum.

The symptoms of chronic otorrhea are usually nothing more than a purulent discharge and an impairment of hearing. Should any pain, vertigo, nausea, nystagmus, headache or facial paralysis be superimposed, the presence of a complicating lesion is usually indicated and the case should be more carefully watched.

In the diagnosis of a chronic suppuration, a careful history of the course of the disease and a local and very often a general medical examination are important. The local examination should not be limited to the diseased ear alone, but an examination of the good ear and a routine survey of nose, throat and sinuses, as well as the dental condition, should always be included.

The character of the discharge is important and should be carefully noted, because it will give information regarding the extent of the disease and the prognosis[18]. The discharge may be scanty or profuse, thin or thick, serous, mucoid or purulent, or an admixture of dried pus, blood, epidermis and cerumen. Some forms emit a fetid odor, while others may be free from odor, or what odor there is quickly disappears after an appropriate local treatment has been instituted. As a rule, an odorless mucoid or mucopurulent discharge indicates a tubotympanic infection due to faulty nasal hygiene; a thin, purulent discharge with a persistent fetid odor means caries or necrosis of bone; a rather thick or cheesy discharge with white flakes and accompanied by a persistent offensive odor suggests the presence of cholesteatoma.

In the otoscopic examination the appearance of the drum head should always be carefully noted, particularly the size and location of the perforation. Some perforations may be so small as to easily escape one's notice if a Siegle otoscope is not used. Others may expose the tympanic cavity and even reveal the structure of the

internal wall of the middle ear. Still others may show only a narrow ring as the remains of the drum head.

A perforation of the anterior portion of the drum head or a combination of an inferior and central perforation may be regarded as favorable insofar as the prognosis and treatment are concerned. Perforation[2] of Shrapnell's membrane and marginal perforation in the posterior superior quadrant are to be looked upon as more serious, for necrosis of the ossicles or cholesteatoma is often associated with these types of perforations.

The presence of chronic otorrhea not only endangers the sense of hearing but there is a possibility for the necrotic process in the middle ear to extend through the tegmen, labyrinth, the lateral sinus or through other portions of the cranial wall, and death may result from purulent meningitis, brain abscess or from pyemic thrombosis of the lateral sinus.[19] The risk is greater in the ignorant than in the intelligent, owing to the fact that the former often have an idea that a discharging ear is a trivial matter and, consequently, it is given little or no attention.

Therefore, in the care of the chronic running ear, one of the important points is to make the patient realize what serious conditions may develop from a discharging ear and why treatment is necessary.

The principle of treating chronic otorrhea is similar to the principle of treating any other disease. The first essential is the elimination of those factors in the nose, throat and nasopharynx which will give rise to an inflammation of the Eustachian tube; especially[3], in children, the removal of diseased tonsils and adenoids should be emphasized. Attention must also be called to the fact that not infrequently adenoid growths recur because of an incomplete removal at the first operation. Such a condition should be carefully searched for and corrected in order to complete the cure and prevent the recurrence of the discharging ear. Of course, dental caries and infection of the nasal sinuses must not be overlooked. The general hygiene of the patient must be also considered. Tonics may be given if necessary to build up the patient's general condition. Injection of autogenous vaccine[4], injection of boiled milk[16] or any specific treatment may be given if indicated.

The treatment of suppurative otitis itself may be generally discussed as local therapy and surgery. Every case of chronic otorrhea does not present the same degree of potentially serious complications and each must be dealt with on its own merits. To distinguish cases requiring local therapy or surgical intervention is important and

difficult[23]. It requires not only one's skill to make a careful examination of the case, but also a sufficient clinical experience to evaluate his observations. Such skill and clinical experience only the specialist in otology can hope to acquire.

Local treatment[13] should, however, be always first instituted and be persisted in in all cases of chronic suppuration where an actual complication is absent. It is only fair to give the patient the opportunity of being relieved of his condition without resorting to surgery. The attempt can do no harm even if it does no good. As a matter of fact, in the treatment of chronic suppurative otitis media, most of the otologists today are inclined more toward conservatism.

Of course, in the local conservative treatment, three important points must be also taken into consideration: First, whether the patient is able and willing to pursue a persistent treatment; second, whether the attending physician is willing to give a careful personal attention to the case; and third, we must have a clear guiding principle as to how long the conservative treatment may be employed.

By surgical treatment we mean mainly an intratympanic operation and the so-called radical mastoid operation.

"An intratympanic operation or ossiculectomy is indicated when a purulent inflammatory process of the middle ear does not respond to a persistent local treatment and in cases in which the disease process is chiefly confined to tympanic membrane, the ossicles, the antrum and the tympanic walls."[24]

"The complete radical mastoid operation is indicated: *1*. when a permanent cessation of the purulent process has not been effected by prolonged local intratympanic treatment, combined, if necessary, with such operations as removal of granulations, enlarging perforations, etc.; *2*. when a cure has not been effected by the removal of the necrosed ossicles and the curettage of the middle ear; *3*. when acute symptoms of mastoiditis supervene in chronic suppurative otitis media; *4*. on the appearance of chills, fever, vertigo, pain or other unusual symptoms; *5*. the appearance of facial paralysis during the course of chronic otitis media purulenta; *6*. attacks of vertigo, nausea and vomiting, indicating that the necrotic process involves the labyrinth; *7*. in all cases of complicating intracranial or lateral sinus involvement; *8*. when there are positive symptoms of cholesteatoma in the mastoid; *9*. when there are fistulous openings in the cortex of the mastoid process or in the osseous canal wall; *10*. whenever extreme depression or any other symptoms of disturbed mentality accompany the disease."[1]

In the local treatment for chronic otorrhea various methods have been discussed in textbooks and literature. Some of the principal

ones may be mentioned, as follows: The so-called "dry wick treatment," irrigation by means of warm saline or warm boric acid solution, aspiration method, the so-called continuous drainage method[21] by means of a well made and elongated swab of highly absorbent cotton frequently inserted into the ear canal, the so-called Calot's[12] solution treatment, the use of antiseptic and dehydrating agents, such as mercurochrome, metaphen, the instillation of ether or boro-alcohol in combination with the instillation of shrinking and antiseptic solution into the nostrils, the insufflation of boric acid powder or the so-called iodin dusting powder (Sulzberger), zinc ionization, etc.

The essayist wishes, however, to discuss particularly the use of iodin dusting powder and the so-called zinc ionization in the treatment of chronic otorrhea.

The so-called iodin dusting powder (Sulzberger) is a patent preparation consisting of boric acid powder, in which there is contained in a finely disseminated state 2 per cent of elementary iodin in the "strong" form and 1 per cent in the "weak" form. The action of the powder[20] is due chiefly to the activity of the free iodin liberated when it is dissolved in the ear discharge.

It has distinct advantages over the ordinary boric acid powder: first, it has a stimulating effect on the injured mucous membrane; second, it has a more powerful antiseptic action; and third, it does not cake as the boric acid powder often does, to form a hindrance to the drainage. Though few articles were written about its use, it is a valuable preparation and should be tried by otologists in the dry method of treatment for chronic running ear.

In this form of treatment[22], it is essential to cleanse the ear canal and the middle ear as thoroughly as possible by means of a small cotton applicator. No irrigation is given. In order to facilitate the action of the powder, granulations, polypi of sufficient size or necrosed ossicles must be removed. When a perforation of the drum is too small, it should be enlarged so that the powder may reach the diseased area. Sometimes gentle suction is necessary to remove some secretion within the middle ear.

The procedure is simple. After the ear canal is carefully cleansed, the powder is insufflated into the middle ear through the perforation. This is accomplished by introducing an insufflator through an ear speculum. For initial treatment, the "weak" form is used. When the discharge does not decrease after a number of treatments, the "strong" form may be given. The treatment is given twice a week and no home treatment is advised. If the secretion is profuse, the patient is instructed to use only clean, dry cotton wipes.

In favorable cases, after only a few treatments, signs of improvement will be noticed. The discharge becomes less and some change in the odor is noticed. In intractable cases there is very little or no sign of improvement after a few weeks or months of treatment. Such cases should be augmented with some other local measure, such as zinc ionization.

The zinc ionization method[6] is not new. It has been a long-standing procedure with dentists, but was not used for the treatment of suppurative otitis media until 1914.

The term "zinc ionization" may be misleading for the mere act of dissolving a zinc salt in water produces an ionization without the passage of an electric current. Therefore, the term "zinc electrosmose" is probably a more scientific expression.

The theory of ionization[11] may be briefly explained as follows: The infecting organisms are viewed as masses of colloidal protein, leukocytes and the colloidal protein possess a negative charge and consequently they are attracted to the anode or positive pole. This is the reason why the positive electrode is placed in the external auditory canal when passing an electric current.

Zinc is used for ionization because it possesses advantageous properties over other metals, namely, copper and iron. Zinc has a higher atomic weight (zinc, 30; copper, 29; iron, 26) and a faster ionic speed (zinc, 89; copper, 47 16) than either of the others.

Experiments[5] have proved that without the aid of an electric current, the action of zinc is only superficial, but with the aid of it the zinc ion can penetrate into the tissues to a sufficient depth to reach bacteria present in the deeper layers.

As to the polarity of the current, it has also been proved that the use of a negative pole in suppurative ear increases the alkalinity of the discharge, making the medium more favorable for the growth of the bacteria. The negative pole is a vasodilator. It increases the discharge, softens tissues and may induce hemorrhage, while the positive pole is a sedative, a vasoconstrictor. It stops hemorrhage and hardens tissues. This is another reason why the positive pole is applied to the discharging ear.

A few words about the types of current used for zinc ionization may be mentioned here. Most writers recommend the straight galvanic current derived from dry cells or a storage battery. But, in 1925, Warwick and Stevenson suggested the use of a direct (Faradic) current generator as the source of an induced electric current for zinc ionization. The principal advantage of using the latter type of current is that it does not so readily produce vertigo or give a

burning sensation as the galvanic current often does. However, the writer has had no experience with it and cannot offer any opinion here.

There is also some difference of opinion among the writers in regard to the addition of glycerin in the zinc solution for ionization. Stevenson explained that, according to Faraday's law, the solution containing the glycerin is only three-fourths as efficient for ionization treatment as a solution of zinc sulphate in water, because the dielectric constant of the two is quite different (the dielectric constant of water is 81, that of glycerin is 39.1). On the other hand, some writers[14] claim that when glycerin is added, the surface tension is reduced and an intimate contact of the mucous membrane and bacteria with the zinc solution is the result. The writer also has not had enough experience to pass any judgment at present.

The procedure and technique of zinc ionization that the writer has employed may be briefly described as follows:

1. *Machine:* On the market there are different electric machines that can be used for ionization purposes, such as the Fisher galvanic current generator, Fisher portable galvanic current generator, battery type, "Electronizer," the Mueller galvanic battery, etc. As a matter of fact, any electric machine in the form of dry cells, a storage battery, or D. C. current from the electric light and power station, that will register about 10 milliamperes with a rheostat or regulator, will serve the purpose. The writer, however, happened to find in the hospital a machine by the tradename of "Fisher low voltage generator" which also generates a straight galvanic current when it is connected with the ordinary electric light socket, with the current selector properly placed. This was employed.

2. *Electrodes:* For the positive pole, the so-called Yerger's ear electrode is used, which contains a metal part and an insulator. The latter is made of hard rubber in the form of an ear speculum. There are three different sizes, all of which will fit into the metal part, which is a half-split spring ring, with a zinc electrode extending to the middle of the speculum when it is connected, and a universal handle to be attached to the cord from the machine. The insulator is to be inserted into the ear canal for protection and also for keeping the zinc solution from running out of the ear canal. But the writer often finds that the insulator is too short and does not fit tightly into the ear canal, so that the metal part will touch the skin of the auricle and the zinc solution often leaks out. To prevent this a short and smooth, soft rubber tube may be used to attach to the end of the insulator. For the negative pole, the so-called Fisher moist-surface electrode pad, 3 x 4 inches, is used.

3. *Solution:* Zinc sulphate, 5 gr. in 1000 c.c. of water with 60 c.c. of glycerin, is kept in a colored bottle and ready for use.

4. *Selection of Patients:*[10, 15] Ionize all cases of chronic otorrhea due chiefly to tympanic or tubotympanic sepsis. Before ionization, any granulations or polypi obstructing the way or preventing the access of the zinc solution into the tympanic cavity are to be removed by means of aural snare or ring curettes under local, or general anesthesia, if necessary. If there is an association of adenoid vegetation, diseased tonsils, dental caries, nasal polypi, septal deformities, diseased nasal accessory sinuses, etc., they are also to be corrected in order to effect the cure and prevent the recurrence of the ear discharge. Those cases that have an attic or marginal perforation are usually unfavorable for ionization, but a persistent ionization, accompanied by an attic irrigation with an attic cannula, should be tried. Those who have mastoid involvement, labyrinthitis, facial paralysis, signs of intracranial complications or bony stenosis of the external ear are not given this treatment.

5. Before application of ionization, the ear canal and the middle ear are to be wiped as clean as possible with small cotton pledgets and followed by irrigation with warm zinc solution (simply by mixing two parts of the prepared zinc solution in one part of warm water). Any air bubbles within the perforation may be removed by means of suction with a Siegle's otoscope.

6. Let the patient lie on side on a table with the affected ear upward. The prepared zinc solution (dilute with equal quantity of warm water) is dropped into the canal with a medicine dropper until the canal is half-filled. A little pressure with a finger is applied on the tragus so as to induce the solution to fill the upper part of the Eustachian tube.

7. The positive cord is attached to the Yerger's electrode, which is simply placed in the ear canal to a depth approximately half the length of the canal, while the negative pole is attached to a flat, moist pad electrode, which is fastened to either arm or leg of the patient by means of a bandage. Add sufficient zinc solution to cover the zinc electrode.

8. Before turning on the current, be sure to have the zinc electrode pretty well submerged in the zinc solution, the skin of the auricle free from the metal part of the electrode, the current selector properly placed and the regulator on the zero mark. Then turn on the current and move the regulator slowly to one milliampere for a minute and then gradually increase to two-three milliamperes. If patient complains of any burning sensation or pain, the amperage

must be reduced until patient is feeling comfortable, usually around two milliamperes. Keep the current running at one milliampere for 30 minutes, two milliamperes for 20 minutes, or three milliamperes for 10 minutes. Near the end of the desired time, very slowly turn the regulator back to the zero mark. Any sudden movement of the regulator will give the patient vertigo and dizziness. Remove the electrode and suck off the excess of solution in the ear with a medicine dropper and let the patient get up.

9. Gently apply dry cotton pledgets in the external canal to remove any excess of the zinc solution but do not disturb any white or grayish-white coagulum, which is often seen deposited around the drum head or within the perforations. Instruct the patient not to put anything in the canal or to irrigate the ear until the next visit. No home treatment is given. Only dry cotton wipes at the external auditory meatus may be used at home if necessary. Patients are often seen twice a week, but ionization is given only once a week.

BRIEF REPORT OF CASES TREATED.

In the ear, nose and throat clinic of the Memorial Hospital, Worcester, Mass., from October, 1930, to May, 1931, there were 31 cases of chronic suppurative otitis media treated by the writer. Eleven cases were treated with ionization treatment, 10 with iodin dusting powder, and two with the combined treatment of zinc ionization and iodin powder; five cases were referred for T. and A. operation, three have not returned after one or two treatments. Most of the cases that were treated with ionization or iodin powder had already had a T. and A. operation.

The duration of the aural discharge of these cases varied from four months to 27 years. Nine had bilateral and 22 unilateral discharge. Two had a radical mastoid operation on one side. Twelve had a mucopurulent discharge with considerable odor. Sixteen had a thin, purulent discharge with fetid odor and three an offensive, scanty discharge with white flakes. Six had granulations or polypi obstructing the canal. Eight had an anterior inferior perforation of the drum, five a central perforation, 14 a perforation of the posterior superior quadrant, three a perforation of Shrapnell's membrane, and one had a large and extensive perforation with only the annulus tympanicus left.

As to the result of the treatment, six have been dry for two to seven weeks after one to six treatments of zinc ionization; three dry from one to four weeks after eight to 20 treatments of iodin dusting powder. The rest had been markedly improving, and are still under treatment.

ILLUSTRATIVE CASES.

*Case 1:* M. L. J., white, female, age 39 years, came to the clinic on March 17, 1931, with a history of running ear of the right side since October, 1930, following grippe. Her chief complaints were profuse ear discharge, impaired hearing on the right and occasional earaches with some sensation of stiffness on the right side of the neck. Patient had been under physician's care most of the time and had been using irrigation and boroalcohol ear drops all along.

Examination revealed that the left ear was negative; nose, nasopharynx and larynx all negative. Tonsils small and mildly diseased. All sinuses transilluminated well. There was profuse, pussy discharge from the right ear. The drum head could not be seen, due to the presence of a large polyp filling the half of the canal. There was no tenderness or periosteal thickening of the mastoid process. Voice test: Whisper, right, two feet; left, 20 feet. Spoken voice, right, 10 feet; left, 30 feet. Tuning fork test: Low limit, right, 256 d. v.; left, 32 d. v. All high notes and Galton's whistle present on both sides. Schwabach was much prolonged on the right and normal on left. Rinné, negative on right and positive on left. Weber, lateralized to the right.

A polyp about the size of a large pea was removed with an aural snare and ring curette under local anesthesia and zinc ionization was given for 15 minutes at 2½ milliamperes. No other treatment was given. One week later on return visit, the patient's right ear was seen dry and clear. Voice test: Whispered voice, right, 18 feet, and tuning fork test, low limit, 64 d. v. Patient was seen on April 21, 1931, and the ear was still dry.

*Case 2:* L. K., white, female, age 30 years, was seen in the clinic on March 24, 1931, complaining of left ear discharge with occasional earaches and impaired hearing. Patient had bilateral running ears since 3 years of age, following scarlet fever. Tonsils and adenoids were removed at the age of four. A radical mastoid operation was done on the right about 14 years ago and is dry, but the left ear discharges practically all the time, and has never ceased. On account of deafness the patient was advised to learn lip-reading three years ago.

Examination revealed some pussy secretion in the left nasal floor. Tonsils removed but some small tonsil remnant seen on right inferior pole. Nasopharynx clear, all sinuses were transilluminated clear. X-ray also showed all sinuses clear, and pronounced sclerosis of the mastoid. The left ear discharge was foul, mucopurulent, canal stiff and narrow. The drum head was dirty and chronically inflamed,

all landmarks indistinct. A fair-sized perforation was noticed over the posterior superior quadrant. Voice test: Whispered voice, right, not heard; left, eight inches. Spoken voice, right, three feet; left, 10 feet. Tuning fork test: Low limit, right, 512 d. v.; left, 256 d. v., faint. All high notes and Galton whistle present on both sides. Both air and bone conduction slightly decreased. Rinné, negative on both side, and Weber, lateralized to left.

The ear was cleansed with cotton pledgets and followed by irrigation of zinc solution. Ionization was given to left ear for 20 minutes at two milliamperes. A week later the left ear was seen to be only moist and nothing running out. Patient could hear better. The left ear was cleaned again as usual and another ionization treatment given for 20 minutes at two milliamperes. Another week later the ear looked dry and clean, with the landmarks of the drum clearly seen. According to the patient's story, she can hear many things that she could not hear before and for the first time she was able to enjoy the sound picture. Voice test: Whispered voice, left, seven feet. Spoken voice, left, 20 feet. Tuning fork test: Low limit, left, 128 d. v. April 28, 1931, about four weeks after the last treatment, the patient's ear was still dry and clean, with the perforation unhealed.

*Case 3:* K. E., white, female, age 13 years, came to clinic on July 18, 1930, complaining of left ear running, on and off, for six years, following measles. Had T. and A. operation at age of seven. Examination revealed that throat and right ear were negative. There was a question of mild ethmoiditis. The sinuses otherwise transilluminated clearly. The left ear discharge was purulent and profuse, with fetid odor. After cleansing, a perforation over the posterior superior quadrant of the drum head was seen. Irrigation with warm boric acid solution, combined with nose drops of ephedrin sulphate gr. V in neosilvol, 10 per cent, was instituted for about two weeks, with some improvement. Owing to the development of some excoriation of skin around the external auditory meatus, patient was advised to discontinue the irrigation but to use dry wipes and ear drops of boroalcohol, in addition to the nose drops. The treatment was continued for over four months and the improvement was very slow. There was still a considerable amount of discharge, with foul odor. Hearing test at this time as follows: Whispered voice, right, 25 feet; left, seven feet. Tuning fork: Low limit, right, 32; left, 256, faint. On Nov. 10, 1930, dry treatment with iodin powder was started and given twice a week regularly and persistently. The improvement seemed to be steady, though slow. On March 22, 1931, the left ear was seen absolutely dry and clean. Patient was seen on

April 17 and the left ear was still dry. Hearing test: Whispered voice, left, 15 feet; low limit, 128; all high notes and Galton whistle were present.

*Case 4:* L. B., white, female, age 15 years, came to the clinic on Dec. 12, 1930, with a history of right ear discharging since four months ago, following grippe. She was treated by the family physician and had used ear drops at home for more than three months. No improvement was seen. Instead, patient often experienced some headaches and dizziness, in addition to gradual loss of hearing. Had T. and A. operation four years ago. Examination revealed that the left ear was normal, nose and throat were essentially negative. Right ear discharge profuse, purulent and foul. A large polyp, fairly firm in consistency, obstructed the canal and the view of the drum head. Hearing test: Whispered voice, left, 25 feet; right, not heard. Spoken voice, right, eight feet. Tuning fork: Low limit, left, 32 d. v.; right, 512 d. v. All other high notes and Galton whistle were present on both sides. Schwabach on right was much prolonged. Rinné, negative, and Weber, lateralized to the right.

The polyp was removed with difficulty under local anesthesia. A large central perforation could be seen, with loss of the handle of the malleus. Insufflation of iodin powder was given for three weeks and little improvement seen. Discharge continued to be profuse, purulent and foul. Zinc ionization treatment was then given for the first time, 20 minutes at two milliamperes. Four days later on return visit, the aural discharge was much decreased, though it still gave a very foul odor. Iodin powder was insufflated again after the ear had been cleansed. Henceforth, the patient was seen regularly twice a week. Ionization was given only once weekly and between the intervals the ear was insufflated with iodin powder. The treatment was thus continued faithfully and regularly for about 3½ months. On April 14, 1931, the right ear was seen perfectly dry and clean, with marked improvement of hearing. Whispered voice heard at 15 feet and low 64 d. v. was also faintly heard. Patient was seen again on April 27 and the ear continued to be dry and clean.

*Case 5:* H. T., white, male, age 15 years, was referred by an otologist on Dec. 19, 1930, for a trial of zinc ionization treatment for bilateral suppurative ear condition, which had continued for about 13 years, following scarlet fever. He has been constantly under the care of specialists and everything was tried, but he never yielded to any form of treatment. Had T. and A. operation at age of six. Because his hearing was becoming worse, he was advised to learn lip-reading during the past three years.

Examination revealed that the discharge of both ears was profuse and purulent, with a fetid odor. A fair-sized marginal perforation was seen on both anterior superior and posterior superior quadrants of the left drum head. The perforation was more or less filled with granulations, while the right drum showed only one large marginal perforation over the posterior superior quadrant, which was also filled with granulations. Hearing test showed whispered voice, right, three feet; left, eight inches. Spoken voice, right, 10 feet, and left, seven feet. Tuning fork: Low limit, right, 256 d. v., faint; left, 512 d. v. All other high notes and Galton whistle were present on both ears. Schwabach was much prolonged on both sides. Rinné, negative, and Weber, no lateralization to either side.

Granulation tissue in both ears was incompletely removed by ring curette at several attempts, followed by the application of silver nitrate, 50 per cent. Zinc ionization was given weekly at two milliamperes, 20 minutes each time. Patient was seen twice weekly and between the intervals both ears were cleansed with dry cotton pledgets and followed by iodin powder insufflation.

The treatment was continued quite regularly for about three months and marked improvement in both discharge and hearing was noticed. According to patient's own words, "there is practically nothing running out" except in the early morning when he gets up. At home he can now hear mother's voice, while in school he does not need to sit in the front row any more. For some reason or other he did not return for treatment for more than three weeks.

On his return for the second time, both ears were seen discharging more and granulation began to appear again. The treatment was started again as before. He was last seen on April 28, 1931. There was still some thin discharge in both ears, with less odor, and some granulation tissue between the perforations of the left drum. Hearing test: Whispered voice, right, 20 feet; left, 15 feet. Tuning fork: Low limit, right, 64 d. v., faint, and left, 128 d. v. He is still under treatment.

In the first case the chronic suppuration was due to a tubotympanic infection, with an association of a polyp. It responded to the ionization treatment beautifully when the polyp was removed.

Case 2 shows that a tympanic infection which is resistant to other forms of local treatment often yields to zinc ionization. Case 3 may show that cases which do not respond to irrigation or boroalcohol should be tried on dry method with iodin dusting powder. The length of treatment might be shortened should ionization be also given. While the last two cases represent those cases in which there

is not only tympanic sepsis but bony necrosis and growth of granulation and polypi are superimposed. They are very resistant to local treatment and will most probably not yield to either iodin powder or ionization alone, so a combined treatment should be persistently tried.

In conclusion, in the treatment of chronic otorrhea, it is not only important to check the diseased process and stop the discharge, but also important to preserve and restore the function of the ear as much as possible. The experience of most otologists tells us that a radical operation on the ear results in most instances in a depression of the hearing. In other words, an improvement of hearing in most cases of chronic suppurative cases can be brought about only by local conservative measures. Therefore, in the absence of an actual complicating lesion, the attempt should always be made to treat all cases of chronic otorrhea conservatively. In the local conservative treatment, time, patience and willingness on the part of both patient and the attending physician are necessary.

In the above report, because of such a small number of cases and short space of time, it is difficult to interpret the results of treatment by zinc ionization, iodin dusting powder, or the combination of both. Nevertheless, the writer believes that they are valuable adjuncts to our local conservative measures for the treatment of chronic otorrhea. The combined use of the two especially may be considered as one of the most valuable therapeutic aids for the treatment of more stubborn cases of chronic suppuration. A persistent treatment should be encouraged as long as there is a steady improvement of the case, with no signs of a complicating lesion.

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## TOOTH DISCHARGED FROM THE NOSE: CASE REPORT.

DR. DOUGLAS MACFARLAN, Philadelphia.

B. A., a young man, age 24 years, had suffered for years from a purulent discharge from the left nostril with occasional neuralgia pains in the region of the left antrum.

When I first saw him, he produced what he said was a piece of bone which he had blown out of his nose. Examination showed the bone to be the first upper left bicuspid, and this tooth was missing from the upper set.

The purulent discharge had ceased and nothing but the normal antra opening was found. Transillumination showed a shadow.

1805 Chestnut Street.

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# TONSILLECTOMY UNDER GENERAL ANESTHESIA IN THE MODIFIED ROSE POSITION.

Dr. Edward F. Ziegelman, San Francisco.

Tonsillectomy has received so much attention by the general practitioner as well as by the laryngologist that any new discussion or presentation of the subject cannot fail to be accompanied by reticence and trepidation.

A very high percentage of tonsillectomies are performed by general practitioners. In all justice to this vogue I must confess that many of the general men do a neater and more skilled surgical operation than some of the men in the specialty. Of course, that does not prove that conditions should remain as they are. It only proves that the special field needs improving as far as tonsillectomy is concerned[1].

Tonsillectomy is considered by many laymen as well as by some in the medical profession to be a procedure of great simplicity, that the last word has been said, and that it is the first step in the chain to the development of a general surgeon, and that economically it is the rent payer.

I know of no method by which the profession is able to obtain honest statistics in regard to the mortality of tonsillectomy *per se*. I am convinced that tonsillectomy possesses one of the highest mortalities in the entire realm of surgery. This belief is based on my personal observation and on deductions drawn from discussions with my professional colleagues, especially general surgeons who are chiefs of general services in open hospitals. In a measure, this is due to the prevalence of the operation, at times also to the lack of concern with which the procedure is done by many and the assumption that it is a simple operation accompanied by slight, if any, mortality.

Frankly, I believe it is one of the most difficult as far as avoidance of complications is concerned. It should be approached with at least an air of responsibility. One of the greatest aids that could befall the medical profession would be to forever forget that empirical classification now so prevalent—the so-called minor and major surgery. All surgery is major. A lacerated finger or a tonsillectomy can be accompanied by as much grief as can a posterior gastroenterostomy. I believe it is imperative that the medical profession

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assert itself against a fallacy that has for its primary basis the tick of the clock. In other words, just because a surgical procedure is completed in 15 minutes, as against an hour for another, is no reason to believe the former is accompanied by less danger, both as to mortality and morbidity. I realize this statement is rather far-fetched. It is made with the purpose of impressing the fact that tonsillectomy should not be looked upon as a surgical procedure unaccompanied at any time by mortality or morbidity.

Technical methods of removal of tonsils vary in a measure, depending upon the custom of the particular locality, though I believe I am safe in saying that the prevalent method is by sharp dissection with the use of the snare for removal. Various knives and snares have been developed. All of these are based upon the method of sharp dissection with the use of the constricting snare. Other methods preferred by many men comprise techniques almost too numerous to mention. Briefly, some of the more popular are the straight

Fig. 1. Surgical tray showing, from left to right: Davis-Sewell mouth gag, tonsil tenaculum, Bard Parker knife with curved blade, scissors, E. Fabré-Rajotte tonsil dissector, pillar retractor, thumb forceps, Gottstein curette, two eight-inch Murphy clamps, Tyding's snare.

scissors dissection and various types of tonsillotomies. These latter seem to have increased in popularity during the past few years. An example of this type of instrument is the Sluder, the La Force or the Beck instrument. A popular technique at present being used and its results being reported is the so-called suction method[2]. Another popular method that recently has become the vogue, especially among neurotic, nervous patients of unlimited means, is the removal with the so-called diathermy apparatus, either with the desiccating or electro-coagulating current[3]. This method of removal by what is known as electrical currents gives promise of becoming very popular and may in great measure supersede the cold knife dissection. Dan McKenzie has been an ardent advocate of this method. He believes that it is superior to any other method we have.

Just as the technique of the removal and the instruments devised vary, so does the position of the patient during the surgical pro-

cedure. Some men, especially those trained under the Boston technique, prefer their patients in a sitting position. This is not the rule, as most operators prefer their patients in the flat position, face upward, the operator standing to the side of the patient's head and performing the tonsillectomy in that position. I am not in accord with either of these positions and will attempt to describe a different one and my reasons for its adoption.

When a tonsillectomy is performed there are certain surgical objectives desired. Briefly these are: first, a complete removal of the tonsillar tissue; second, the least trauma to the tissues; third, the avoidance, as far as possible, of bleeding; and fourth, the prevention of complications. The first three of these objectives are so

Fig. 2. Photograph of position of patient while undergoing the anesthetic.

well recognized and so involve the fundamentals of surgical principles that it would be useless to discuss them. The question of complications is recognized by many but it seems to be the rock upon which catastrophes occur. Consequently, I shall confine my remarks to the fourth objective desired—the avoidance of complications.

This resolves itself into two important questions: first, the prevention of secondary hemorrhages; and second, the prevention as far as possible of lung complications; in other words, pulmonary abscesses. I know that some of my confreres will say that such a discussion as this is theoretical and far-fetched, based on the absence of these complications in the practice of some men. From numerous reports of excellent and reliable men[4 to 19], I believe it can be claimed

safely that pulmonary abscess is not a mythical complication of a tonsillectomy, especially of a tonsillectomy performed under general anesthesia. Anyone who has spent much time in hospitals must have seen what were evidently post-tonsillectomy abscesses. When we fail to see them, we fail to look for them and also fail to analyze our postoperative conditions carefully, justly and scientifically. I think that every case of postoperative pneumonia or persistent bronchitis, especially of the purulent type, should be investigated from the standpoint of a pulmonary abscess. If we are willing to assume that such conditions do occur, then it seems reasonable to attempt to overcome them by all means possible. I shall discuss briefly these two complications that are to be avoided: secondary hemorrhage and

Fig. 3. Photograph of so-called modified Rose position, mouth gag in situ. Anesthetist with left hand controlling mouth gag.

pulmonary abscess, and then describe a technique which I believe lowers to the minimum the incidence of these complications.

Most secondary hemorrhages are controllable with a sponge. It is true that at times such a condition occurs as to tax the patience, fortitude and skill of the operator[20]. Such hemorrhages usually occur at the base of the tongue from the so-called lingual area and necessitate the use of a ligature to control. Practically all secondary hemorrhages of the tonsils are avoidable by a proper ligation at the time of the operation. It is surprising how few tonsillectomies are primarily ligated. The usual prevalent method is to sponge until dry or to trust to hemostasis by the use of slowly constricting snare. We are all agreed that in most instances such technique is sufficient,

though not always, as may be vouched for by many laryngologists. I believe that the lack of use of the primary ligature is due to the difficulty with which it is applied. This difficulty is due in a great measure to the position of the patient on the table. It can be overcome by use of the technique I am about to describe.

Lung abscess, the other complication to be avoided, is a comparatively new surgical entity[21]. Either pneumonia, purulent bronchitis or gangrene of the lung were the names given to it in days gone by. Only with the advent of the bronchoscope and the development of the Roentgen ray[22] has the incidence of this complication been both appreciated and determined. We are all certain it occurs. As to its method of development, two schools hold sway at present, one

Fig. 4. Left tonsil has been dissected free. Note removal with snare and tenaculum. Hands not crossed.

favoring the so-called aspiration method[7, 11], the other the so-called circulatory method[1, 23, 24]. Both groups seem to have excellent experimental and scientific proof of their opinions. I am inclined to believe that both methods are correct and that an abscess of the lung may develop by either of the two courses. Although I am convinced that some abscesses of the lung occur by way of the circulation, I am also inclined to believe that, as far as the laryngologist is concerned, the prevalent method is by way of aspiration. The avoidance of trauma with the use of a clean dissection and a slow constricting snare in the position I shall later describe not only lessens, in my opinion, the possibility of aspiration but makes the development by way of a circulatory invasion almost a minimum.

That portion of the pharynx common for both respiration and deglutition extends from the soft palate above to the cricoid cartilage below where it becomes continuous with the esophagus posteriorly and with the larynx anteriorly. This common pharyngeal area is further subdivided into an upper oral and a lower laryngeal portion. This arbitrary line of division is the hyoid bone. The oral portion opens anteriorly through the faucial isthmus into the mouth; the laryngeal portion, extending from the hyoid bone, continues into the esophagus. Anteriorly, it has the triangular entrance into the larynx with its valve-like structure, the epiglottis. On either side of the laryngeal orifice, extending downward for some distance, are the pyriform sinuses or pouches.

Fig. 5. Same as Fig. 4, only manipulations reversed in the removal of the right tonsil.

The six muscles of the pharynx, three constrictors with the stylopharyngeus, salpingopharyngeus and palatopharyngeus, are intimately concerned with the physiology of swallowing. I believe they are important factors in the aspiration of foreign material into the larynx, trachea, bronchi and lungs during operative procedures under general anesthesia. One of the great difficulties encountered in pathological lesions of the pharynx involving these muscles, either myopathic or neurogenic, peripheral or central, is the inability to avoid aspiration of foreign materials into the respiratory passages.

The question of the type of anesthesia as well as of the condition of the patient during anesthesia in the consideration of the depth of the narcosis has always been of great interest to both the anes-

thetist and the surgeon. It is important to the life of the patient and to the ease with which the technical procedure can be accomplished. Still, I believe altogether too much importance has been placed upon the anesthetic side of tonsillectomy when it comes to the analyzing of conditions of the lungs. One school insists that deep narcosis leads to the formation of abscess of the lung; the other, that light anesthesia is a sure precursor of that development. I believe that both are correct as long as they are willing to operate with the patient in either a sitting or a horizontal position.

Under deep narcosis the reflexes are abolished, the constrictors do not function. Blood that gets into the hypopharynx and pyriform fossa, after filling these spaces, gravitates mostly into the esophagus

Fig. 6. Removal of adenoids with the Gottstein curette.

and to a less degree into the larynx. These deductions are based upon bronchoscopic and esophagoscopic examination during the performance of tonsillectomy under general anesthesia by innumerable observers[25]. Anyone who has made these observations is convinced that at times a portion of the blood gets into the larynx and trachea.

If one is willing to concede that secondary hemorrhage is at times a fact, that abscess of the lung does occur and that blood gets into the larynx and bronchi at times during and following a tonsillectomy under general anesthesia, then it is fair to assume that a technique that will to a great extent overcome these difficulties is not out of place, both as to description and acceptance (see Figs. 1-6).

In the technique which I wish to describe briefly, the patient is in the so-called modified Rose position, well known to those who

do plastic surgery about the mouth. It is as follows: The operating table, on which the patient lies flat on his back, is lowered 30 degrees below horizontal. Any type of mouth gag is used. I prefer the Davis-Sewell. The operator sits at the head of the patient. When the mouth gag is opened the entire hypopharynx comes into view. A small sponge is placed in the nasopharynx to avoid blood getting into that space. Then the tonsils are removed, according to any technique desired. I prefer to use sharp dissection with slowly constricting snare. All bleeding is controlled by ligature, a complete view of the hypopharynx is had and of the pyriform fossa. In fact, one can always see the epiglottis during the operation. After the tonsils are removed the sponge is removed from the nasopharynx, then the adenoids are removed, using any type of curette desired. I prefer the Gottstein. While doing this, a sponge is placed in the hypopharynx to catch and absorb any blood that may gravitate downward during the adenoid operation. After the removal of the adenoids with the curette, a final inspection of the nasopharynx is made with a piece of gauze on the index finger. Any adenoid tags are removed in this manner. I consider this matter of precaution against retention of remnants of adenoid tissue a very essential part of the technique. One can easily inspect the fossa of Rosenmüller in this manner. Finally, a complete inspection of the tonsillar area is made, a sponge previously soaked in 50 per cent alcohol, then wrung dry, is gently pressed in each tonsillar fossa, as well as in the nasopharynx, and the patient is returned to bed. For 12 hours after operation the patient remains in prone position in bed.

The advantages of this technique are: one has complete direct view of the entire operating area; it is the logical position in which to tie vessels that may be spurting, especially in the lingual region. The position overcomes the gravitation of blood into the pyriform sinuses, pharynx or larynx, thereby overcoming the so-called aspiration of material with the formation of abscess in the lung. Any disadvantage, such as that sometimes claimed that the blood gravitates into the nasopharynx, with danger to sinuses or Eustachian tubes, is overcome by the use of the sponge in the nasopharynx during the operation. To my knowledge, I have never had a lung, Eustachian tube or sinus complication since I adopted this technique.

## CONCLUSIONS.

*1.* Aspiration is apparently one of the causes of abscess of the lung.

*2.* Position of the patient during operation is probably a factor in the formation of aspiration.

3. The so-called modified Rose position from the standpoint of mechanics overcomes the tendency to aspiration.

4. This position is ideal for orientation of the operative field.

5. It is the logical position for the control of hemorrhage in the prevalent and always difficult area—the lingual area.

6. It is the position of choice for the prevention of surgical shock.

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## LIVING FOREIGN BODIES IN THE LARYNX.

DR. F. ROBBINS, New York.

Living foreign bodies, in the form of insects, carried by the wind, may enter the air passages, especially of speeding automobilists, by way of the mouth or the nose. As a rule, such visitors promptly die and are expelled by forcible coughing or sneezing, but in other cases are aspirated and retained in the larynx, usually in the epiglottic space or in the ventricle of Morgagni, occasionally penetrating as far as the bronchi. This article is not concerned with accidental intruders of this kind, nor does it deal with the small inhabitants of aquariums, tadpoles or sticklebacks, sometimes encountered in the throat of "eat 'em alive" artists. There are certain relatively large animal parasites which survive and have their being in the larynx and its immediate environment, being essentially represented by: *Echinococci, ascarides, trichinae*, and last but not least, *leeches*.

With special reference to the *echinococcus*, Manciolo's case, published in a rather inaccessible Italian periodical, is of such interest as to be entitled to reproduction: The patient, a man of thirty-one years, with a negative family and personal history, came under observation on account of a swelling in the neck and expulsion of numerous cysts from the mouth on the night before admission. For over two years, he had been troubled by mild attacks of sore throat and hoarseness. These attacks increased in frequency and about one year ago, a somewhat protuberant tumefaction appeared in the right side of the neck, which on palpation appeared to be about the size of a small egg. This tumefaction increased parallel with disturbances on the part of the fauces, and a distressing sensation of weight supervened in the chest, especially during the night, and in the dorsal position. The voice had become considerably altered, but the dysphonia was much less marked when the patient turned the head towards the right instead of the left side. On account of increasing oppression of the chest, the patient caused a linseed poultice to be applied to the swelling on the neck; soon afterwards, a high fever supervened and persisted the entire day; at night, while resting on the right side, without need of coughing, he felt an irritation in the throat as if due to a foreign body, with regurgitation to the mouth. Soon afterwards, he successively passed from the mouth, four large cysts filled with

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fluid, the size of an egg yolk. The expulsion of entire, smaller cysts continued on the next following days, so that in the course of three days, altogether about forty cysts were voided. Some cysts were also voided with the stools. At the same time, the tumefaction in the neck, which had finally reached the size of half an orange, had become considerably diminished. Well-marked dysphonia was present. All the expelled cysts were entirely intact, and at first sight could be recognized as echinococcus cysts. The examination of the cyst fluid and easy demonstration of the characteristic hooklets confirmed the already clear diagnosis.

On objective examination, palpation on the right side of the neck showed a round, smooth, slightly movable tumor, not adherent to the cutaneous tissues, and which in the act of swallowing became displaced together with the larynx. There was nothing of importance on the part of the oral and pharyngeal cavity, but the patient's breath was extremely fetid and offensive, a condition which according to his statements had begun to manifest itself only four or five days ago. Extraordinary importance is attached by the observer to the laryngeal examination: It could be noted that the right arytenoid region was markedly hypertrophied and composed of a flap of reddened mucosa with lobulated margins, movable on respiratory movements. On manual compression of the tumefaction on the neck, a large quantity of a yellowish extremely offensive fluid could be seen to flow from the subjacent region to this flap of mucosa. The form of this movable flap suggested its constitution by the mucosa which lined the inferior surface of the loosened and detached arytenoid. The unusual condition in this case accordingly concerned a large echinococcus cyst, developing in the right lobe of the thyroid and opening spontaneously on the lower surface of the right arytenoid, raising and detaching the regional mucosa. The patient was operated upon and a rightsided thyroidectomy was performed. The thyroid was found to be adherent to the larynx and communicated with the laryngeal interior through a tract the size of a steel pen. On opening the extirpated lobe of the thyroid after its removal, it was found to be replaced to a large part by a cystic sac with roughened walls, over half a centimeter thick towards the external surface, tapering off towards the laryngeal surface; the cyst still contained numerous extremely small daughter cysts. Following the operation, the patient was attacked by severe bronchopneumonia, evidently due to ingestion of food, but at the end of a month had completely recovered. The laryngoscopical examination showed total paralysis of the vocal cord on the right side, an extremely common event in thyroidectomy. The segment of mucous

membrane constituting the arytenoid region was strongly retracted and shortened, in its interior. At the time of the report, five months after the operative intervention, the patient was in good health and since four months had resumed his work on the railway. Manciolo reports this unusual observation as the first case of echinococcus cyst of the thyroid, rare in itself, opening into the larynx. (Atti della Clinica OtoRinoLaringologica della R. Universita di Roma, 3:109-111., 1905.)

In the Mercedes Hospital in Havana, Cuba, about twenty years ago, the writer saw a case of echinococcus cyst of the thyroid gland, which had perforated into the larynx, through the annular cartilage, as in an old case described by Boch. The most salient disturbances in this patient, a consumptive woman about sixty years of age, consisted in vocal cord paralysis and aphonia, through pressure on the recurrent nerve, which had been misinterpreted as due to laryngeal tuberculosis.

An extremely rare localization of the echinococcus parasite in the close vicinity of the larynx was observed by O. Langemak, in 1925. The patient, a woman age 75 years, some weeks before coming under observation noticed a protuberance at the posterior pharyngeal wall, which caused no pains, but gave rise to slight swallowing disturbances. Examination showed a cystic tumor, attached by a broad base to the posterior pharyngeal wall, adherent to the mucosa. Puncture yielded a turbid serous fluid which aroused a suspicion of echinococcus. The treatment was operative, with incision of the rather immovable mucosa over the tumor. Incision of a connective tissue membrane brought into view a whitish glistening membrane which could not be extracted as a whole but only piecemeal, and on microscopical examination was found to be a chitin membrane. The patient recovered after open wound treatment without drainage.

*Ascarides* not uncommonly, as a result of their migration into the stomach, are then expelled through vomiting and thus may reach the upper air passages. The penetration of ascarides into the larynx and into the trachea is especially dangerous, because it is liable to lead not only to severe attacks of dyspnea, but even to sudden asphyxiation. Oppolzer (1844) observed a fatal case through the penetration of a worm into the larynx. Wagner more recently (1902) observed a boy of eight years, in whose case a convolution of ascarides was vomited from the stomach and blocked the laryngeal entrance leading to death from asphyxiation. Fuerst in 1879 compiled twenty-five observations on the immigration of ascarides in the larynx and trachea. In Mosler's patient with aphonia and dyspnea, the parasite was successfully

removed from the larynx. Donati, in Italy (1878) noted four ascarides in the larynx, and Cerchex (1891) observed asphyxia and aphonia through the presence of ascarides in the larynx or in the trachea, respectively. Wegresco's patient, a child of three years, died from asphyxia due to an ascaris which had reached the larynx and from there the trachea. The observation of Landa, in Russia (1909), concerned a girl of three years, suffering from pneumonia; a few days after the onset of the pneumonia, a convolution of ascarides appeared in the mouth, followed by asphyxia and death. The autopsy findings showed that the mouth, pharynx and larynx were filled with ascarides. In the case of a child who had died under symptoms of acute laryngeal stenosis, Zolatkowski (1908) found that after death, a mass of ascarides which had blocked the laryngeal entrance, emerged from the throat. In the experience of Rabot (1904), a child suffering from diphtheria was not relieved by tracheotomy and did not breathe freely until a worm appeared in the cannula and was removed from it.

Another important animal parasite is represented by the *trichina spiralis;* for the larynx no less than the buccal cavity, the pharynx and nasopharynx may participate in general trichinosis, and in some instances, the muscular tissue of the larynx has been found to be especially strongly interspersed by trichinae. The associated inflammatory phenomena, in the form of congestion and edema, lead to disturbances of motility through a typical myopathic vocal cord paralysis. Following the encapsulation of the trichinae, the paralysis of the affected laryngeal muscle is apt to prove permanent, although not always total, and mild cases may terminate in complete recovery. Paresis of the vocal cord tensor muscles was observed by Seifert in a woman who two years previously had suffered from acute trichinosis and during this time was completely aphonic.

Some illustrative cases were reported by J. E. Mackenty, in 1908, who calls special attention to the involvement of the upper respiratory passages in trichinosis infection. The condition observed in the throat and larynx of his patients did not show an acute surface inflammation, but looked more like passive edema. In two of his cases, the throat symptoms predominated, and in the first case, concerning a woman of sixty-eight years, he was actually called to do an intubation for a gradually increasing laryngeal stenosis. "The laryngeal mirror showed edema extending down the lateral walls and over the epiglottis. The edema in the upper laryngeal region completely obscured all view of the part below." Her daughter, a woman of thirty-two years, was the second patient, and also in this case, the larynx showed

edema of the arytenoids and false cords. The true cords were scarcely distinguishable from the surrounding swollen tissue. These observations are highly instructive and may well serve to widen the diagnostician's horizon in doubtful or obscure cases. As pointed out by Mackenty, the diagnosis in sporadic cases is very difficult. The prognosis depends upon the gravity of the general trichinosis. His patients apparently suffered from acute trichinosis. Special interest is attached to the detailed observations of Navratil, in Budapest, on a rare chronic case of laryngeal paralysis as a sequel of infection with the trichina spiralis, eighteen years before the patient entered the clinic. The emaciated anemic woman, forty-four years of age, who appeared much older than her actual age, suffered from paralysis of the vocal cords, in the presence of unrecognized chronic trichinosis. On examination, the left half of the epiglottis was found to be immovable, while the right half regularly exerted its function during respiration and phonation. The vocal cord and arycartilage on the left side were immovably fixed in the median position between respiration and phonation. Although the right vocal cord as well as the right arycartilage reached the median line during phonation, the movements of both, especially the tension of the vocal cord, showed a certain degree of sluggishness. In consequence of the leftsided laryngeal paralysis, the closure of the glottis was incomplete and the voice was muffled. The patient complained of dyspnea, which had recently increased in severity, threatening asphyxiation on mounting upstairs. The diagnosis had to be left open for the time being, in view of the misproportion between the objective and subjective findings. The condition did not undergo the slightest change during her six weeks' sojourn in the clinic. Some months later, she was seen again in worse condition than before. The same phenomena were noted as at the time of her first admission, with total laryngeal paralysis on the left side, sluggish motion on the right side. Swallowing and breathing were said to be more difficult than before. The local and general condition from now on became rapidly worse, and during the last eight days, the swallowing disturbances reached such a high degree that she was hardly able to partake of any food. The patient finally succumbed to rightsided lobar pneumonia. The autopsy findings furnished no information as to the reason of the persistent paralysis of the epiglottis and larynx; but the microscopical examination of the laryngeal muscles proved more instructive: Even with the unaided eye, numerous hard, light, elliptic points were visible in the laryngeal muscles and under the miscroscope were shown to be calcified trichina capsules, situated in the muscle fibres. With the exception of the heart and the intrinsic muscles of the tongue, the

entire striped muscle tissue showed the same behavior. Each capsule contained a trichina, in which certain movements could be elicited on heating the specimen. The number of trichinae was greater in the leftsided crico-arytenoid posticus muscle than in the same muscle of the right side; in the two thyreo-arytenoid muscles, the trichinae were present in approximately equal number. The chronic infection in this patient dated back to a sojourn in Galicia, eighteen years ago. The observer points out that this case not only widens the etiology of laryngeal paralysis by a new factor, but lends additional support to the diagnosis of chronic trichinosis, an especially grave form of this infection. Following calcification, the muscular contraction encounters a mechanical obstacle through the calcified trichina capsules between the sarcolemma and the muscle fibre, resulting in atony, paresis or paralysis of the affected muscle, according to the number of existing capsules. This assumption was confirmed in the foregoing case by the patho-histological findings, according to which the left-living patient, was densely interspersed with trichina capsules, while in the corresponding muscle on the right side, which merely presented atony of a less degree, the calcified capsules were considerably less numerous. Paralysis was first and constantly observed in the laryngeal and ocular muscles, which represent a site of predilection for the trichinae, and which owing to their small size could offer less resistance against the calcified capsules than the pharyngeal constrictors, which actually did not become paralyzed until later on. In the presence of laryngeal paralysis, the direct or remote cause of which cannot be discovered, especially when also elsewhere, for example in the ocular muscles, paralysis (squinting) is present without the affected individual having previous'y suffered from the same, there is sufficient reason, in view of the eventual presence of general muscular atrophy, to suspect an infection with trichina spiralis and to proceed to biopsy.

*Leeches:* A recent observation on living foreign body in the larynx, in the form of a leech, was contributed by a Japanese writer, Kayno Maruyiama, in the Oto-Rhino-Laryngological Department of the Medical Faculty of Taihoku (Formosa). The patient was a man of fifty-six years, who began to complain of paresthesia of the throat immediately after drinking river water. On examination, a moving leech was discovered in the patient's larynx and was extracted by way of direct laryngoscopy. The animal measured 10 cm. in length. (Otologia, Fukuoka, 3:228., 1930.) A parallel observation concerned a case of apparent hemoptysis, due to the presence of a leech in the larynx, seen by Maurice Yoel in the Oto-Rhino-Laryngological Serv-

ice of the Piraeus Evangelical Hospital in Athens. This leech had reached the larynx at the time of the patient's drinking from a brook and had caused hematemesis during one month. By means of laryngoscopy, the leech was finally discovered below the vocal cords, as a black body the length of the little finger, and was extracted with forceps. (Archiv. Internat. de Laryngol., 8:1198., 1929.)

The older literature contains numerous observations on accidents due to the presence of leeches in the air passages. In three cases observed by Broc, in France (1910), the leeches were attached at the laryngeal surface of the epiglottis. An instructive observation by Zografides (1913) concerned a hunter of 40 years, who had slept in the woods and on the next day complained of irritation in the throat, cough and swallowing disturbances. The patient was declared to be tuberculous by his physician, but laryngological examination showed a leech in the depth of the larynx, with a subglottic situation of the head. On attempting to grasp the leech with the forceps, it slipped into the trachea; severe dyspnea supervened and tracheoscopy showed that the leech had attached itself to the wall of the trachea, but it could be easily pulled off and extracted with forceps.

A leech was removed instrumentally, after cocain anesthesia, by Zakopal (1917) from the larynx of a soldier who a fortnight previously had drunk water from a brook, in Asia Minor, and since that time had repeatedly expectorated blood. In three Turkish soldiers, who were admitted with a statement of "worm in the throat," and one of whom suffered from severe dyspnea, Haerting (1916) found leeches in the larynx, attached to the vocal cords. In a case observed by Faine, in France (1913), the leech was situated in the subglottic space. Tonietti, in the Tripolis campaign, observed fifteen cases of leeches in the upper air passages and learned of four other cases from his colleagues; in five of these cases, the leech was lodged in the larynx. In four cases reported by Aboulker in Algiers (1914), the seat of the leech was found at the laryngeal entrance. Gaggia, in Italy, in the Libyan campaign (1914), saw twenty cases of leeches in the upper air passages, including eighteen of the larynx, all with a successful outcome. In a fatal case reported by Blandin, the autopsy findings showed that a leech obstructing the rima glottidis had induced death through sudden asphyxiation. Seven cases of large horse leeches in the larynx, trachea and nasopharynx, were reported by Citelli, in Italy (1912). The four cases concerning the larynx were recognized and treated with the assistance of the laryngeal speculum:

1. Woman of about fifty years, who for ten days had passed blood with the sputum and feared that the trouble was due to a leech.

Endoscopy showed the posterior wall of the pharynx to be covered with partly red, partly clotted blood. Towards the stem of the epiglottis, slightly above the false vocal cords, a relatively small blackish body was attached, evidently a young horse leech. It was at once extracted with Fraenkel's laryngeal forceps and the patient made a complete recovery.

*2.* A male adult had passed blood from the throat since ten days and from time to time complained of attacks of dyspnea. The pharynx contained partly fluid and partly clotted blood, and in the larynx a large leech was seen, lying over the vocal cords. On extraction, it was found to be a large horse leech, attached to the right false vocal cord. The patient was immediately relieved of all disturbances.

*3.* Another patient, a woman, for a fortnight had expectorated blood and suffered from some attacks of moderately severe dyspnea, as well as intermittent dysphonia. A large leech was found attached to the lower portion of the arytenoid region. The leech when distended, reached as far as the subglottic region: this caused the alteration of the voice and also to a large part the attacks of dyspnea. The foreign body was at once extracted and was found to be a horse leech.

*4.* A young man of twenty years had raised blood for ten days and suspected the presence of a leech. The pharynx contained red and black blood. In the larynx, a horse leech of moderate size was found, which after extraction could be shown to have been attached on the right false vocal cord.

The foregoing observations show that large as well as small leeches may penetrate into the air passages by means of the drinking water. The large leeches are less movable and therefore less likely to reach the bronchi, but they may more easily, in the larynx or at the beginning of the trachea, give rise through their large size to alterations of the voice and respiratory disturbances, besides causing a considerable loss of blood for the bearer. The respiratory disturbances are in part permanent and due to the mechanical factor of the presence, near or within the rima glottidis, of the body of the leech and sometimes also a blood clot. In part, these respiratory disturbances occur in attacks, being due to reflex laryngeal spasm.

*Treatment:* The general practitioner, when confronted with a foreign body in the upper respiratory passages, may proceed as follows: Foreign bodies in the larynx and pharynx can often be removed with forceps or sometimes with the fingers, under guidance of the laryngoscope. In the presence of asphyxia, the performance of tracheotomy or cricotomy, respectively, is indicated. Haphazard exploration with forceps, sounds or catheters must be cautioned against. If unavoid-

able, a delay of even a few days, and transportation of the patient to a specialist department, is much less dangerous. In the management of individual cases, importance is attached to the anamnesis, the subjective behavior, the general condition, the findings on auscultation and radiography. Surgical interventions, for example tracheotomy, have their own special indications and must be left to the specialist. In consequence of infection with the trichina, some or all muscles of the larynx may become involved, the resulting symptoms pointing either to a peripheral muscular affection, disease of the nerve trunk, or sometimes even to a central cause.

The addition of a complete bibliography would consume too much space and is therefore omitted. The main source of references can be found in Denker-Kahler's Diseases of the Throat, Nose and Ear, Part 4., 1928 and under the respective headings in the Index-Catalogue of the Library of the Surgeon General's Office, U. S. Army. Live fishes, impacted in the air passages, larynx, trachei and bronchi, all these organs being treated as one, have been reported, in 1926, by E. W. Gudger, Ph. D., Bibliographer and Associate in Ichthyology, American Museum of Natural History, New York.

2 East 103rd Street.

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## CONTROL OF PAIN FOLLOWING TONSILLECTOMY.

DR. ALEXANDER F. LASZLO, New York.

While a tonsillectomy may be performed painlessly by the experienced surgeon by means of anesthetics and skillful technic, the problem of rendering the convalescence agreeable is nevertheless one which still involves considerable difficulty. True, the patient may be primarily concerned with a painless operative procedure. However, the suffering which he is subject to for several days after the anesthesia has disappeared is of sufficient magnitude to warrant serious consideration on the part of the physician.

It is generally conceded that the younger the patient, the less he suffers. Thus children up to five or six years of age seem to have little trouble partaking of a regular meal one day after the operation. It has also been observed that an operation performed under a general anesthetic is apt to cause less post-operative pain than what is inflicted under local anesthetic. This phenomenon may be due to the

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injection damaging the tissue and causing additional pain, although it is frequently attributed to the fact that the general anesthetic causes the sensory reflexes to be retarded for some time after the operation. There is today a growing tendency towards removing tonsils in adults under local anesthesia and we are here primarily concerned with easing pain following this specific method of operating.

Skillful operative technic, gentle handling of the tissues, will lessen the pain. Similarly, less mutilation of the pillars, less injury to the muscular tissues behind the capsule contribute considerably towards a more agreeable convalescence. Hemorrhage in the uvula with its resulting edema, is one of the most painful sequels to a tonsillectomy.

The pain will usually start within a half hour to two hours after the operation, depending upon the type of anesthetic used, and is severest during the ensuing twenty-four hours. It usually radiates to the ear.

The treatments employed to alleviate post-operative pain vary greatly, each operator having his own method. Seedy, Herzig and Trotter advocate the use of quinin urea hydrocloride, asserting that lc.c. injected into the nerve on each side will eliminate the pain for a few days after the operation. However, this method never became popular for several good reasons. First, if the injection does not reach the nerve fibers the operation is quite painful. Secondly, it is necessary to wait 15 to 20 minutes for the solution to take full effect. Thirdly, quinin urea occasionally causes sloughing. Nupercain (Ciba) is alleged to possess lasting local anesthetic effect. While it works quite well at times, in many cases the anesthesia lasts for a brief period only. Hyperdermic injections of opiates such as morphin, codein and pantopon are still resorted to. These are, in my opinion, wholly unnecessary. The use of orthoform or anesthesin powders is necessarily limited as the method of administering these drugs calls for trained hands. More popular are these same drugs in the form of lozenges, or aspirin in the form of powder or chewing gum. An ice collar will also ease the pain.

It is, of course, difficult to obtain the true conception as regards the merits of a drug whose beneficial action necessarily must be appraised by the patient. It is equally obvious that in subjecting such a preparation to a test great care must be exercised in selecting patients whose judgment merits consideration. It is a well known fact that the hypersensitive patient is apt to suffer most.

Recently my attention was called to a drug distributed under the name of "Euphagin Tablets" originated by Dr. Haslinger of the Hajek Klinik in Vienna. These tablets contain amidobenzo acidethy-

lester, some menthol and some sodium borate. They are non-toxic. About eight to ten minutes is required to dissolve the tablet in the mouth. I was much impressed with their powerful anesthetic action in trying them on myself. The saliva becomes impregnated and should be allowed to trickle into the fossa. The anesthesia is present within a minute after the tablet is placed on the tongue and persists for about an hour. The surface sensibility is considerably lessened but the anesthesia does not penetrate sufficiently deep to materially affect the muscular reflexes.

We have been using Euphagin tablets routinely in the operative clinic combined with aspirin chewing-gum, as the exercising of the jaws in many cases speeds up the convalescence. No opiates are ever prescribed, not even codein. We have also used Euphagin successfully in a few cases of other painful mouth and throat afflictions such as carcimona, pemphigus, stomatitis and ulcerations of the mouth and throat. In addition to its anesthetic action, the sodium borate and menthol contained in these tablets will assure antiseptic effect. No untoward effects have been observed from the use of Euphagin tablets, which therefore may be used uninterruptedly during the first few days following tonsillectomy.

In conclusion I may add that Euphagin is used to advantage in Esophagoscopy and has been found a valuable adjunct to cocain in Laryngoscopy and Bronchoscopy.

124 East 84th Street.

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## AMERICAN BOARD OF OTOLARYNGOLOGY.

An examination was held in Indianapolis, Ind., Sept. 12, 1931, just prior to the meeting of the American Academy of Ophthalmology and Otolaryngology held in French Lick, Ind. Forty-three candidates were examined, of which nine were conditioned or failed.

The Board will hold an examination in New Orleans on May 9, during the meeting of the American Medical Association, and in Montreal next fall, just prior to the session of the American Academy of Ophthalmology and Otolaryngology.

Prospective applicants for certificates should address the Secretary, Dr. W. P. Wherry, 1500 Medical Arts building, Omaha, Neb., for proper application blanks.

H. P. Mosher, M.D., *President.*

W. P. Wherry, M.D., *Secretary-Treasurer.*

## International Digest of Current Otolaryngology.

*Editor:*
Dr. Maxwell Fineberg, St. Louis.

*Collaborators:*
Prof. G. Bilancioni, Rome.
Mr. W. S. Daggett, London.
Priv. Doz. G. Kelemen, Budapest.
Dr. H. C. Rosenberger, Cleveland.

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Professor Georges Portmann will conduct an intensive course in otolaryngology in Washington, D. C., Feb. 1 to 6, 1932. Inquiries concerning this course can be addressed to Dr. James A. Flynn, 1511 Rhode Island avenue, N.W., Washington, D. C.

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A new Westinghouse three-element X-ray tube and machine makes possible the taking of an X-ray snapshot in less than 1/1000th of a second. This enables physicians to see clearly, for positive diagnosis, the inside of a human body; up to this time blurred or hidden in X-rays by the motion of the patient's muscles. This new instrument was shown for the first time at the convention of the Radiological Society of North America at St. Louis.

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Claude L. LaRue, of Shreveport, in the Sept. 26, 1931, *Journal A. M. A.*, presents an article on "Laryngology in Relation to the Hematopoietic System." This difficult subject is discussed in a clear, terse manner, and the author comments on purpura and agranulocytosis. The laryngologist may be among the first to see these conditions and unless he uses the laboratory for a study of the blood he undoubtedly will fall down on the diagnosis of these conditions.

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Bednar and Langfelder, of Sillein, Slovakia, in the *Monatschft. for O. L. R.*, present an original article on "Intravenous Stimulation of the Organ of Smell." They conducted careful experiments and came to the following conclusions: *a.* Certain substances produce individual smelling reactions when introduced into the body by the intravenous method. *b.* Between the beginning of the injection and the beginning of the reaction, there exists a definite latent period which accounts for the time necessary for the injected stimulant to reach the olfactory region. *c.* Through paralysis of the nerves of smell, or through atrophy of the mucous membranes which are affected

by anosmia, there is no hematogenous stimulation while the hematogenous smelling power in respiratory anosmia remains intact. *d*. Hematogenous smell reactions are maintained through intramucous stimulation of the endings of the olfactory nerve.

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Cohen and Rudolph, of Cleveland, in the October 3, 1931, issue of the *Journal A. M. A.,* presents an interesting article on allergic and infectious conditions of the upper respiratory tract in children. The subject is very ably presented and of great interest to the oto laryngologist is a table of differential diagnosis between allergic and infectious conditions of the upper respiratory tract. The table is divided into three sections: History, Examination and Treatment. This chart in itself will be of value as a reference in the attempt to make a diagnosis in difficult cases.

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There is an editorial in the September 19, 1931, issue of the *Journal A. M. A.,* on the Electro-Surgical Removal of Tonsils. The editorial stresses the importance of choice of case for electro-coagulation. Much has recently been written about this method and it is undoubtedly gaining popularity in the hands of the general practitioner, the surgeon and the borderline specialist. Manufacturers of the instruments are partially to blame for the unwarranted popularity because of their excessive claims of advantages. Tonsillectomy should still be considered a major surgical operation and in competent hands, in indicated cases, there is no doubt but that electro-coagulation is of service; however, the feeling prevalent today among the recognized specialists is that electro-coagulation is merely another valuable adjunct to the armament used in tonsillectomy.

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George B. Bader, of New York, in the Sept. 26, 1931, *Journal A. M. A.,* presents a case report on acute suppurative parotitis with spontaneous rupture into the external auditory canal occurring as a complication to mumps.

A three-year-old child with acute purulent otitis media of the right ear developed a swelling at the angle of the jaw on the left side which soon extended around to involve the entire left side of the face. History of mumps in the family some two or three weeks before. Swelling on left side of face was incised, at which time it was noted that there was a sagging of the superior canal wall which ruptured spontaneously. The course of the disease was uneventful and there was no residual paralysis of the face. The author believes that this condition occurs more frequently than the literature indicates.

## THE NEW YORK ACADEMY OF MEDICINE.

SECTION OF LARYNGOLOGY.

*Meeting of Feb. 25, 1931.*

**Symposium On Esophagus: Diverticula. Dr. Frank H. Lahey (by invitation).**

DISCUSSION.

DR. A. C. CRUMP: I think that both of our societies feel extremely pleased over the wonderful presentation given us this evening. The esophagus itself is one of the blind spots in our anatomy for diagnosis, largely through its poor sensory nerve supply, difficulty in swallowing being generally the initial symptom met with in most cases.

Carcinoma of the esophagus is the most important disease, in my mind, that has to be dealt with in this organ and an intensive study by our two societies can do a lot by proper co-operation in arriving at an early diagnosis. Jackson has pointed out that "puddling" in the pyriform sinus is one of the earliest symptoms met with in these cases. When carcinoma has reached the stage of ulceration it is then too late for surgery, X-ray or radium to be of any use. Paralysis of a vocal cord may also point to carcinoma of the esophagus before difficulty in swallowing occurs.

Diseases of the esophagus are comparatively rare. On a combined admission of about 500,000 cases in 10 years in the Presbyterian and Bellevue Hospitals, there were only 665 cases of disease of this organ; a little over 0.2 per cent. Of these, over 50 per cent were carcinoma; cardiospasm, 4 per cent; stricture, 7 per cent; and diverticula, 3 per cent.

As to Dr. Lahey's paper, I wish to congratulate him on his excellent work. He has certainly simplified and rendered much safer a dangerous operation.

There are a few points that I might add toward aiding in the diagnosis and treatment of diverticulae of the pulsation type. A discussion as to its origin is nominal. Lawrence Abel, of London, places the site of Zenker's diverticula above the point given by Dr. Lahey. He gives the location as a small triangular space above the cricoid muscle and below the fibres of the interior constrictors, which the lantern slides will show you.

In considering the treatment, the first and second type that Dr. Lahey spoke of can be helped a great deal by simple means. The obstruction met with in swallowing is frequently caused by inflammation in the sac, resulting in zonal spasm. In these types a lavage of the sac at bedtime to get rid of the fermenting food will frequently result in the patient again swallowing without difficulty.

Lavage is also beneficial in the third type. Posture in a rather unusual position—face down, head below the shoulders and over the edge of the bed—will frequently enable the patient to swallow food, to their surprise. The patient is instructed to first empty the sac by pressure on the side of the neck. Food is then given them through a gruel tube, which they swallow up hill, so to speak. The mechanical principle is simple. Air occupies the empty sac and when a bolus of food is swallowed this air is compressed, so naturally the food takes the direction of least resistance—the esophagus. The mechanical action is similar to the air tank on a fire engine. This method is useful in placing a thread guide into the esophagus before esophagoscopy to aid in finding the esophageal opening.

Regarding cicatricial stricture, Dr. Tucker has discussed this subject beautifully and his retrograde treatment can hardly be improved upon; however, I differ with him in some particulars.

In the examination of these cases strictures situated at the introitus are extremely difficult to see as the distal end of the esophagoscope cannot displace the larynx sufficiently to see into the region below the cricoid. For that reason

you are blind below this point. The silk thread method is difficult and can be dangerous. The use of the "flexible-tipped" piano wire method as a guide is safe and easily and quickly done (see Blumer's Bedside Diagnosis, Diseases of the Esophagus, Vol. 1, p. 663).

Blind bouginage of the esophagus should certainly be condemned. By blind, however, I do not mean the esophagoscope should be always used. The esophagoscopist does not know what condition exists beyond this point of vision. Palpation beyond this point, unless done on a guide, is dangerous. Bouginage when done properly is of extreme importance in diagnosis when done by one experienced in its use. Fluoroscopy should always precede any instrumental examination of the esophagus.

In treating stricture my dilatations are always done on a "piano wire" guide.

As to the time when dilatation should be commenced, "Dr. Tucker says two weeks." In my opinion, it is only when the acute esophagitis has subsided, which may be a month or a month and a half. An early gastrostomy where feeding is necessary.

Regarding cardiospasm, Dr. Imperatori has given you fully the theories of its cause, so I will show you the X-ray plate of a patient with cardiospasm of the cardia, which is only 12 inches from the incisor teeth. This plate is that of a patient of hernia of the stomachh through the diaphragm—short esophagus type. Mosher's idea and Jackson's idea certainly play no part. It is a spasm of the true cardiac sphincter.

As for the treatment of cardiospasm, I prefer a mechanical dilator as one can feel what is being done. I find X-ray control of the instrument unreliable as the point of the cardia is very variable; it can be moved as much as four inches.

Lavage is of extreme importance both before and after dilatation on account at first of the esophagitis and afterward to prevent food stasis in the esophagus. I will be glad to show my stomach tube and bulb, which I use for this, after the meeting.

DR. JOHN E. MACKENTY: This meeting tonight is the most inspiring I have attended for a long time. The papers have all been excellent and delivered by masters in their own fields. I am not competent to discuss the papers of Dr. Imperatori or Dr. Tucker. But I wish to affirm that their patience and ingenuity in this work is inspiring, and that the advances made in esophageal therapy in the past 10 or 15 years is remarkable. The only part of the symposium I have experience in is that presented so ably by Dr. Lahey.

I must first of all thank him for the pleasure I have derived from his clear, concise treatise on esophageal pulsion diverticulum and for pointing out the several pitfalls and the causes thereof in the seemingly simple yet often difficult operation for its cure. I know of no more grateful patients than those relieved of this most troublesome and, in the long run, fatal disease. In nine years spent in the dissection room, between 1888 and 1902, I observed two instances of traction diverticula occurring close to the tracheal bifurcation on the anterolateral wall of the esophagus. These were funnel-shaped projections of all the layers of the organ.

I also ran across six instances of pulsion diverticula—four on the left and two on the right posterolateral wall of the esophagus. One of these was enormous, stretching into the superior mediastinum and capable on distention of holding eight to 10 ounces of fluid. Three were large enough to project beyond the surface of the neck when dissected out, and two were about the size of walnuts. It is interesting to note that nothing has been added to the true nature and causation of this deformity since the classic treatise on the subject by Sir Charles Bell, 116 years ago. My early experiences were with the one-stage operation. The sac was dissected down to its neck, mattress sutures applied around the neck, the sac cut off, the edges touched with carbolic acid and alcohol, as we used to do in appendectomy, and the stump inverted. The neck wound was packed and left wide open. There was always some infection, but no deaths from this cause.

In only a very few of the diverticula operated was the sac long enough to reach the surface of the neck; yet these small diverticula were giving considerable trouble and demanded excision.

My two-stage procedure then consisted in anchoring the sac as high as possible to some convenient muscle edge with fine silver wire, the ends being left to protrude from the wound. The wound was packed and left open. Eight or ten days later the sac was located by means of the silver wire and removed, as just described.

In the very small diverticula I still adhered to the one-stage operation, fearing the difficulty of again so well defining the sac at the second operation. In one, I inverted a small sac into the esophagus without opening it. One old man died of hemorrhage from the mediastinum seven days after operation following the removal of the largest diverticulum I have ever seen. On removal and when stretched it was seven inches long.

One patient with the one-stage operation developed a very troublesome granulating wound, which took weeks to heal (due to infection).

Two of my series occurred in males under 30 years of age, both patients of Dr. Gage, of Utica. No strictures have occurred. I use a long incision. In one case a very large thyroid made the exposure difficult. I do not close the wound.

The implantation of the sac without tension is not as easy as it might seem, due to the rapid contraction of the sac when detached. A liberal allowance should be made for this.

Dr. Lahey's stressing of points is fundamental: 1. The thorough liberation of the sac. 2. Its high implantation. 3. Implantation without tension. 4. Excision one-half inch or more distal to the esophageal wall.

In long finger-like diverticula it is sometimes difficult to distinguish the sac from the esophagus, and the small bulbous diverticula are sometimes hard to locate. It is here that the aid of a bronchoscopist is invaluable. With the bronchoscope in the sac, orientation is made easy. When the bronchoscope is used to locate the sac it is, before being withdrawn, shifted into the esophagus and a feeding tube is inserted into the esophagus to about six inches below the sac, or half-way to the stomach. The bronchoscope is then withdrawn and the proximal end of the feeding tube is brought through the nose from the nasopharynx forward. In my laryngectomy work I have been using this half-way-to-the-stomach tube for 25 years, with absolutely no untoward effects.

This winter I was in consultation at a diverticulum operation on a feeble old man. The bronchoscopist failed to find the esophageal opening. The surgeon would not consent to placing a tube in the sac, as Dr. Lahey so ingeniously advises. A gastrostomy was done, and the patient died.

I prefer ether anesthesia if there are no contraindications; and I use regional rather than paravertebral anesthesia. The latter does not, in my experience, reach the retrotrachea area; it requires more solution than regional to accomplish anesthesia, and there is always the haunting thought that the needle may enter the spinal space. With scopolamin 1/200 and morphin ¼, regional anesthesia can be satisfactorily carried out in any region of the neck.

I believe that the technique presented by Dr. Lahey tonight is not only the last word on this operation, but perhaps the final word. When possible to do, the two-stage operation is unquestionably the best.

I wish again to thank Dr. Lahey and the Section for the privilege of discussing his paper.

DR. M. C. MEYERSON: I wish to express my appreciation of this very fine program and of the opportunity to hear the splendid presentations this evening. I should like to ask Dr. Lahey whether he uses a nasal feeding tube, and how much time elapses before he permits his patients to swallow. I have seen six cases of pulsion diverticulum. I recall that I had considerable difficulty with each case that I tried to esophagoscope. I, therefore, learned to follow the suggestion of Sippy, to have the patient swallow a string as a guide for the esophagoscopic tube. I believe that the assistance of an esophagoscope to push the diverticulum out of the depth of the neck and into the surface of the wound is of great value to the surgeon who is not doing much of this work. When one is doing as much neck surgery as Dr. Lahey, such assistance would be unnecessary.

Regarding stricture of the esophagus, as you know, the type of stricture will depend upon the penetration of the caustic or corrosive substance which has

been swallowed, the length of the contact, and the point of its contact. If only the epithelial lining is affected, a membranous stricture will result. If, however, the submucosa and muscularis are involved, the stricture will be deeper and more extensive. These strictures become fibrous after a time; in some cases fibrous bands are found stretching across the lumen of the esophagus. Ossification has been known to occur in some of these fibrotic areas. It is these fibrotic strictures of long-standing which are accompanied by dilatation sometimes of considerable extent above the point of stenosis. The esophagoscope enables us to find these fibrous bands, which we can cut, enabling us to later dilate the stricture. Strictures are found single or multiple, complete or incomplete. In children who have a relatively small swallowing passage, strictures are more frequently found at the upper end of the esophagus, because the injurious substance stops there first. In adults, and particularly those who are habitual drinkers, the ingested fluid might pass right down through the esophagus into the stomach without making contact with the esophageal walls. But quite frequently it is observed that the corrosive substance has stopped at the lower end in adults and has caused a temporary spasm, resulting in retention of the injurious substance long enough to occasion severe damage.

I was pleased to hear Dr. Tucker say that he uses esophagoscopic bouginage only in cases of single stricture. I am a strong advocate of peroral bouginage being done over a previously swallowed string as a guide. If the string can be swallowed for retrograde bouginage it can be swallowed and utilized for peroral bouginage in a given case. Dr. Tucker has so perfected his technique that it is a very successful procedure in his hands. My few experiences with gastrostomized patients upon whom I used retrograde bouginage has given me the impression that the procedure is quite painful. I should like to have Dr. Tucker's word on this phase of his technique.

I wish to congratulate Dr. Imperatori for having brought up to date and perfected the technique of the treatment of cardiospasm. The most interesting part of the subject of cardiospasm is its etiology; this is so because of the many theories that have been advanced as its cause. It is of interest to note that Dr. Einhorn, who is here this evening, first propounded the theory of achalasia, a failure of the lower end of the esophagus to open. He is quoted by Hurst, who is today the most active proponent of this theory. I have been following the technique of Vinson, of the Mayo Clinic, in cardiospasm. He has recently to a great extent eliminated the necessity for the use of the hydrostatic dilator. Instead of this, he uses a No. 60 French bougie and obtains very good results in many cases. Dr. Imperatori has a very fine technique, which he has perfected after a good deal of application and study. Because I do not use his technique and do think so much of it, I shall hereafter refer my patients to him if simple bouginage with the No. 60 F. does not help them.

I would like to mention the case of a 2½-year-old child, who swallowed lye and was subjected to blind bouginage. The result was that a mediastinal suppuration resulted, which finally caused a fatal termination.

Dr. Frank H. Lahey: This has been a very interesting evening to me and I have learned some things about stricture and cardiospasm that I did not know before. I have little to add regarding esophageal diverticula. The subject is simple from the point of view of diagnosis. The operative procedures that we have laid down are pretty definite. With due respect to Dr. Tucker, who sees these cases in Philadelphia, I would say that I do not feel it is at all necessary to introduce the bronchoscope into the esophagus in order to visualize them. With the patient in the proper position and the esophagus exposed, it is never difficult to visualize even the smallest.

With regard to feeding, that can be done immediately. The fact that you dissect the sac out and implant it high makes it possible for them to swallow when they get downstairs and go on a full diet immediately. The mucosa is intact and the canal is packed with boric gauze.

Dr. Gabriel Tucker: I wish to thank Dr. MacKenty, Dr. Crump and Dr. Myerson for their discussions of my part in the symposium; also Dr. Lahey and Dr. Imperatori for their kind references to my presentation. I feel that I have learned a great deal about the esophagus from all the men who have

entered into the symposium. I should like to congratulate Dr. Crump on his excellent result with his wire-guided method of dilatation in the case he presented. I believe the same case could have been successfully treated by Dr. Jackson's method of peroral esophagoscopic bouginage. The retrograde method is reserved for those cases where peroral methods have failed to produce a cure as rapidly as is desired, or where a gastrostomy has already been done. After gastrostomy, it is our experience that the stricture can be dilated much more rapidly and safely by retrograde than by peroral methods. In answering Dr. Myerson's question, the child to whom I referred had an eccentric stricture at the hiatal level, not a tubular stricture. The hiatal and abdominal esophagus can be dilated much more safely and rapidly from below than from above; especially if the stricture extends below the level of the diaphragm. A number of fatal cases have been reported from attempts at dilating a stricture at the level of the hiatus from above. On account of this danger, it is necessary to proceed very cautiously with dilatation from above. In regard to gastrostomy, in my cases it has been done low and to the left of the median line so as to enable easy access to the hiatal esophagus. The gastrostomy wound epithelializes usually after two or three months, then as the bougies are used in larger sizes as the esophagus dilates, a larger sized gastrostomy tube is placed in the wound. This prevents undue stretching of the gastrostomy wound at the time of the retrograde dilatation and also the pain that may be produced by dilatation of the gastrostomy wound. The gastrostomy tube, of course, is changed daily. After dilatation is complete, the gastrostomy tube is gradually reduced in size but the opening is maintained for six months or so after the patient is well, to take care of any emergency that may arise. Allowing the wound to close gradually, it usually closes spontaneously and it is very seldom that plastic surgery is necessary. I have observed no ill effects from the prolonged wearing of the gastrostomy tube.

**Symposium On Esophagus: Strictures of the Esophagus.** Dr. Gabriel Tucker.

*(Published in full in* THE LARYNGOSCOPE, *June, 1931.)*

**Symposium On Esophagus: Cardiospasms.** Dr. Chas. J. Imperatori.

*(Published in full in* THE LARYNGOSCOPE, *June, 1931.)*

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## RHINOSCLEROMA.

At the International Congress of Oto-Rhino-Laryngology, at Copenhagen, it was decided that the spread of rhinoscleroma beyond its former geographic limits has rendered desirable a world-wide registration of all cases as a basis for a report on the subject at the next congress in Madrid in 1932. Registration blanks (one required for each case) may be obtained from the Chairman of the Subcommittee, Chevalier Jackson, Temple University, Philadelphia.

# THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

POSTGRADUATE SESSION.

*Regular Meeting of May 5, 1931.*

**Hard-of-Hearing School Child.** Dr. Robert Taylor.

*(Published in full in* THE LARYNGOSCOPE, *November, 1931.)*

DISCUSSION.

DR. ENGLAND: We have just listened to an excellent paper dealing with one of the most important problems, both from a humane and economic standpoint, with which the medical profession is faced today. Dr. Taylor has concisely outlined the method of handling the problem of "what we shall do with the deafened child." I would like to add a few remarks as to the cause and prevention of this condition, which has not received sufficient attention from the profession in general.

There is a growing tendency among the outstanding otologists to disregard the old classification of nerve and conductive deafness, for the following reasons:

1. It is extremely rare to find a case of nerve deafness without some element of conductive deafness.

2. Most cases of conductive deafness develop some nerve deafness or central deafness, which gives findings similar to nerve deafness.

3. The deafened person who comes to you is interested not in his type of deafness, but rather in what you can do for his affliction. Recently I heard "that grand old man," Dr. Gleason, say that when confronted with a case of deafness your chief problem was to determine the percentage loss of hearing, then politzerize or catheterize, and if the hearing improves it is safe to assume that you are dealing with a case of conductive deafness and can tell your patient that he can expect some reasonable amount of improvement. All otologists agree that there is little that can be done for an established nerve deafness, and even conductive deafness does not show the consistent dramatic improvement reported by the enthusiasts of Eustachian bougies, filiforms and applicators. The point I am trying to make is that we are apt to lose ourselves in a mass of scientific data, forgetting that we have a deafened patient, whose hearing we cannot fully restore, and usually, at the best, can only prevent from further progression.

This leads us to the logical conclusion that if we face the problem squarely we must strike at the cause of this deafness. The great majority of these patients will give a history of acute suppurative otitis media earlier in life if you will take the time to question them carefully. It has been proven beyond doubt that even one attack of acute suppurative otitis media can cause a 6 to 9 per cent loss of hearing and, furthermore, that the loss of hearing progresses in proportion to the length of time the ear discharges if it is allowed to go on into a chronic state. The chief causes of acute suppurative otitis media are measles, scarlet fever, influenza and the common colds. That tonsils, and especially enlarged and diseased adenoids, play a major rolé in its development is thoroughly understood, yet we are too prone to forget that this may be the determining factor in whether these patients will clear up in a few days or go on into the chronic state. Herein lies our chief field of usefulness: Early well performed tonsil and adenoid operations, with especial attention to clearing out the Eustachian tube mouths and not being lulled into a false sense of security by removing the central mass of adenoid tissue, which is so often done. A close co-operation between the pediatrician and the otologist. Education of the family in the relationship between deafness and diseased tonsils and adenoids and last, but not least, the treatment of that "bugbear" of all otology, the chronic running ear.

During this year I have had the opportunity to watch the treatment of chronic suppurative otitis media, conservative, operative and postoperative, in

three of the largest hospitals in the city. It has been most interesting to observe the results being obtained, the time they operate after the acute attack, the thoroughness with which the operation was done, the number of postoperative dry ears, intracranial complications and length of time required to secure dry ears after a mastoidectomy. To a plebian observer the following conclusions are quite evident:

1. That the greatest cause of deafness is acute and chronic suppurative otitis media.

2. That far too much valuable hearing is being sacrificed by conservative treatment, allowing these cases to reach a point where only a radical mastoidectomy is indicated, the results of which are certainly not satisfactory from a standpoint of restoring hearing.

3. That the so-called simple mastoidectomy in many hands is only an antrotomy and that the statement that when you drain the antrum your case will get well is wholly wrong from the standpoint of preservation of hearing.

4. That there is a direct relationship between the thoroughness in which a simple mastoidectomy is done and the number of postoperative dry ears and the time required to secure those dry ears after operation.

5. That the early complete simple mastoidectomy offers the greatest hope in securing dry ears and the preservation of hearing.

DR. MACFARLAN: Dr. Taylor's paper is excellent. Its thorough understanding of the subject shows what is appearing everywhere—a growing knowledge and interest in deafness.

Five years ago not many otologists appreciated the fact that there was a handicap to the school child who had a small hearing loss. Testing methods were such that these children were called dull, and not deafened. It is reasonable to suppose that there are as many children with under-functioning ears as with under-functioning eyes. The cost to the community in the aggregate is enormous, for nearly all of these children repeat one or more school terms. Further than this, they grow up with a reduced efficiency that is always a pull on them and on the community. Left untreated, their deafness progresses to a point where it is obvious they must do something about it, but it is often too late. Neither they nor the doctors have the patience to work them out of their trouble. Many cases, no matter how much care is given, will not recover hearing.

We are now conducting a clinic in the graduate school where our particular desire is to get the cases early and to avoid all this future distress. Not these alone, but the advanced or established cases of deafness need attention.

Few otologists realize the terrible strain that these cases have in trying to hear and in trying to keep up socially and economically with the rest of the world.

Something can be done for the advanced deaf beyond lip-reading and hearing aids, and this lies in readjusting them psychologically.

Dr. England's discussion is valuably complimentary to the main paper. Every statement he makes should be emphasized. The question naturally arises, how long shall we let an ear run?

The parent will want to make a try to dry the ear up. Douching offers little prospect. Building the patient up offers more. But telling the parent that an operation may be necessary if the ear does not shortly dry, will get the best co-operation from them, and will prepare the way for the acceptance of the operation. Two months is late enough to delay.

What Dr. England says about incomplete operations and antrotomy is true. The operation is not complete when a few of the accessible cells are removed —let us say, all of the soft bone removed—and the antrum opened. Overhanging edges are left; soft tissue tags are not trimmed, and cells remain. Often the antrum is merely exposed, or it is not made into a good, wide drainage canal.

The matter of postoperative packing is important. It is a basic principle that a packing should be a drain and not a plug.

What Dr. England says about adenectomy is most important. As a routine procedure of the tonsil and adenoid operation the tube mouths should be firmly wiped out with gauze. An adentome should not be used in these low lateral regions.

There has not been mentioned the nasal discharge and the use of ephedrin and silver albummates. All measles cases should have this treatment instituted as soon as diagnosis is made.

I am beginning to believe that postnasal drip is actually the cause of most adenoid and tonsil hyperplasia.

To drain the ear down the tube it is essential to attend to this postnasal drip, for it swells the tube mouths shut. Pus in the middle ear will run out of the ear until it can run out of the tube.

We do not use politzerization enough in acute otitis, with or without perforation. Nor do we use it enough in chronic O. M. P.

There is no reason why we should not train mothers to inflate.

The chief feature of all our thought tonight should be—let us not allow the incipient case to grow severely deaf; let us not think that douching an ear is anything more than cleaning the external ear canal.

DR. STOUT: Dr. Macfarlan said, "Do your submucous even if your tonsils are infected." Sometimes if you do that you have a very severe tonsillitis with other complications following. Think a little bit before you do a submucous if the tonsils are infected. It is better to take out the tonsils first.

Dr. Taylor mentioned that the rich have only 2 per cent deafness. We have not found it so. In one prep school, where only wealthy children can afford to attend, several hundred youngsters were examined and the deafness was over 9 per cent. The deafness of college students is somewhere around 12 to 15 per cent. Finances and care may not be everything. Heredity, accidents and illness are very important factors. Deafness is found as nearly as often in the wealthy as in the poor.

You can do much to save hearing if you get the case in time. Dr. England mentioned about the complete operation. That is a very big factor. Do not stop with a simple mastoidectomy; do a modified radical. Follow this up with treatments for a year or more and you will find that you have saved the hearing and, as a rule, the discharge discontinues, or at least becomes much less.

DR. LEEBRON: It seems to me that when 3,000,000 children in the United States are suffering from partial or total deafness, it is time to look for an explanation. Are we examining the school children thoroughly, is the first question that would naturally arise. It would appear that a society of this nature should be the one to stress the necessity of regular otological examinations in schools in order to detect the hard-of-hearing child and to direct proper treatment which will permanently improve and frequently cure many cases. In the pediatric department of the graduate hospital, we frequently come across children who are backward in school; some of them are even suspected of feeble-mindedness. When we refer them to the otolaryngological department and deafness is discovered, corrective measures, such as removal of physical obstacles, as enlarged tonsils and adenoids, and special training is instituted. These children improve and get along much better in school afterwards. I am of the opinion that if more otolaryngological examinations would be carried on more of these children suffering from partial deafness would benefit by corrections.

There are other conditions causing deafness. They are acute catarrhal inflammations; ethmoiditis and middle ear infections are the most common ones in children. These if not attended to may cause the temporary or partial deafness to progress into a more serious condition.

DR. TAYLOR (in closing): In closing I might say that in some communities proper examination of school children is impossible, due to the cost of the audiometer. I think there would be a difficulty in getting the authorities to purchase this apparatus.

**Laryngeal Stenosis; Its Treatment. Case Report.** Dr. Julian de Armas.

*(Published in full in* THE LARYNGOSCOPE, *November, 1931.)*

DISCUSSION.

DR. TUCKER: It is interesting to note that during this treatment, which covered about one year's hospitalization, there were 78 sittings requiring an aggregate of 58 minutes of actual treatment time. As an explanation of the

result, it would seem that Nature tends to restore the larynx to normal, and that the repeated gentle dilatations through the laryngoscope overcomes the tendency of scar tissue to retract. The end-result in this case is a normal lumen through the stenosed area in the larynx, with an excellent vocal result.

The use of a dilating apparatus worn constantly could not produce this result in the same way. The insertion of a dilating apparatus by means of laryngostomy in children under 6 years of age is a very difficult undertaking and is unsuccessful in many cases because of the difficulty in adjusting and maintaining the apparatus in the proper position. If, during the manipulation or the dilatation by means of an apparatus, these patients develop a perichondritis or chondritis, the treatment is to be stopped during this time, and if the reaction is serious a necrosis of the cartilage may result and the stenosis be increased. As Dr. de Armas has emphasized, the first requisite in the treatment of chronic stenosis of the larynx is a properly placed tracheotomy tube. The next important factor is maintaining the airway through the larynx. In most instances this can be done by repeated gentle dilatations through the laryngoscope. If direct dilatation will not give sufficient airway to permit decannulation, it is usually best to keep up the treatment and when the patient is older a laryngostomy can be done with a much better chance of a good final result.

DR. CLERF: In this excellent report, the plan of treatment for cicatricial stenosis of the larynx has been set forth and a beautiful result has been the term secured. I believe that "high tracheotomy" should be deleted from medical literature. There are some who believe high or low tracheotomy is optional. There should be but one form of tracheotomy and that should be low.

DR. LUKENS: The thing we always stress is early treatment. Several years ago, I had occasion to do some of this work at the municipal hospital. The early cases responded very well. Those that were of long duration did not respond well at all. Success in treating laryngeal stenosis by dilatation depends upon the promptness with which the procedure is begun following the cause.

**The Relation of Dental Infections to Maxillary Sinusitis.** Dr. Victor R. Alfaro.

*(Published in full in* THE LARYNGOSCOPE, *December, 1931.)*

DISCUSSION.

DR. MELOY: Dr. Alfaro's excellent paper so thoroughly covers the subject that a discussion by one of such meager experience as myself seems futile.

There is, however, one point I would like to emphasize. The dental profession is divided into two schools of thought. The first contends that when the dentist opens into the antrum while removing a tooth, he should remove as much of the diseased tissue as possible; then irrigate the antrum, and continue with frequent irrigations through the alveolar wound, making no definite attempt to close it. After several months of this treatment, if the fistula is still draining, a plastic closure is done and the antrum irrigation through the nose, either by the dentist or a rhinologist to whom the patient has been referred.

The second school believes that when the dentist is unfortunate enough to open into the antrum he should clean out the diseased area as carefully as possible and immediately close the wound tightly. Experience with many such cases has shown that if the perforation is small and no meddlesome trauma inflicted, the patient will experience little or no antral discomfort; if, however, there are symptoms of antral infection the patient is immediately referred to a rhinologist for further treatment. The consensus of opinion being that a fistulous tract through the alveolus is maintained not only because of irritation by the food particles which are constantly forced through into the antrum during mastication.

As further argument in favor of the second school, I will cite 11 cases; first, a series of five, in which the antrums were accidentally opened during extractions. Following immediate suture no ill effects were noted. Three other cases after immediate closure developed acute empyema of the antrum, which condition promptly subsided after intranasal treatment by a competent rhinologist. Lastly, three cases in which immediate closure was not attempted but

which were followed by antral empyema. These patients were subjected to frequent irrigations through the alveolar fistuli for from three to four months. During this interval, no amelioration of symptoms could be noted. These fistuli were then excised and the wounds closed by the classical plastic operation. In two of these cases, simple window operations beneath the inferior turbinates sufficed to bring about prompt recovery. The third was cured only after a radical operation.

While there have undoubtedly been many cases in which alveolar fistuli have healed spontaneously following drainage of an antral empyema, still a careful search of the literature and personal contact with dentists of high standing shows such cases to be in the minority.

DR. WEIDER: I think Dr. Alfaro's paper is of extremely great importance, mainly because of one thing. We all know the general prevalence of dental infection, but it is peculiar that many rhinologists are not awake to its importance. I have been astounded to find how few men use dental transillumination. This is one of the most important parts of the examination for every new case. It is absolutely essential to go right through the whole gamut in every case. One of these things is dental infections, which are certainly of great etiological importance in cases of allergic conditions and some sinus infections. While the infection in the mandible may not be responsible for antral disease, the chronic infection lowers the resistance of the patient and whatever slight tendency there is toward infection is magnified. I cannot impress too much the importance of routine transillumination of the teeth.

DR. MACFARLAN: When transillumination came into vogue some years ago, I found, with others, that the tissue around certain teeth showed shadows. Often the teeth were apparently normal, and without a history of trouble. It was a question as to whether soft tissue changes were occurring before bony changes appeared on X-ray. In following up these cases with the dentist there was found no correlation between these transillumination shadows and future dental trouble. Certainly, more often than not, transillumination did not lead the way toward finding dental trouble present at the time. I think that we should leave these matters to the dentist.

The persistence of sinus shadows must be constantly kept in mind; former antrum infections leave thickened mucosa, and yet all the subjective signs of antrum trouble are past. If we keep puncturing these cases we are apt to set up a neuritis. Without symptoms and with no systemic effects from facial infection, it would seem best to let well enough alone.

DR. M. S. ERSNER: I have been extremely fortunate in observing and treating a number of maxillary sinusitis due to dental infection. I have an advantage over other sinusologists, because of the fact that my brother is an oral surgeon and is associated with me at the office. Whether it is due to the fact that we see these cases together or that certain groups of cases are referred to him because of their dental origin, we find that 25 per cent of the cases of maxillary sinusitis are due to dental infections.

We have observed that in a great many cases the floor of the antrum has been torn. A greater proportion of these patients have bleeding from the nose. I believe that it is best to leave it alone, as Nature makes some provision whereby something must happen to the hematoma in the antrum. It seems that after the patients were traumatized, some organisms were forced up into the antrum. Sometimes it becomes necessary to wash out the meatus. This may require from two to six washings, until the return is clear. Occasionally we wash out a small root. We have had a case like the picture you saw several years ago; a part of a tooth was pushed up into the soft tissues and a chronic suppuration resulted. Upon X-ray it was noted that the tooth was lying in the soft tissues.

In 1928, in my paper entitled, "Clinical Observations of X-ray Diagnosis of Maxillary Sinus Disease After the Injection of Lipiodol and Deductions from the Pathological Reports of 150 Radical Maxillary Sinus Operations," I called attention to the relationship between rhinology and dentistry. In some of the large antra the teeth grow very close together. Just the other day I had a case where one of the canine teeth was practically in the antrum. It is easy to understand how infection can take place in a case of this nature.